101 Clinical Cases in Emergency Room

Badar M Zaheer

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Emergency Room

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101 Clinical Cases in Emergency Room

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Dedicated to

The memory of my father Dr Abdul Hafeez who used to say "Be an asset to your community, society, and nation at large and make a difference in somebody's life".
I'll consider myself fortunate to follow in your footsteps.

Preface

"If I cannot do great things, I can do small things in a great way"

— Martin Luther King Jr

Emergency medicine is a budding specialty in the developing world, many people die because of lack of basic emergency services. Some of these injuries or disasters are easily preventable and treatable. Doctors in emergency rooms need to have procedural skills and a broad-based knowledge to treat these emergencies based on the ABCD (Airway, Breathing, Circulation, Deformity) priorities.

Our focus will be to address the immediate life-threatening problems in a quick and efficient way and not to waste valuable time in reaching a final diagnosis.

This book follows the above principles and it is designed to be read by all health care providers who are involved in Emergency Medicine Care including: physicians, medical students, and nursing professionals. It is unique in the sense that it is compiled by a single author speaking from his own 25 years of experience in the ER, combined with up-to-date universal fundamental principles of emergency medicine.

The book is divided into 21 chapters and sectioned by the body system. Tables, charts, and easy illustrations are used to clarify the content and facilitate the learning process. Standard National Treatment Guidelines are stressed in each case discussion. The first golden hour of opportunity rule is stressed in many emergencies including: stroke and MI management, to optimize care and the patient's quality of life. The last chapter deals with disaster management, triaging techniques, and discussing the use of mock trials to train health care workers to manage a mass casualty incident in an efficient way. It will additionally discuss drownings, burns from fire, chemicals, and electrical sources, all of which are preventable injuries. The chapters also stress the need to coordination between governmental law enforcement and the community to help prevent these disasters from ever occurring.

Each case study is designed to stimulate a thought process, especially for students, to formulate a diagnosis and a quick treatment plan accordingly. Clinical Pearls and Pitfalls are provided to keep the importance in mind

of missing a diagnosis as well as the legal implications that would follow. Some historical cases are mentioned to make the subject interesting, easy to remember, and relates to the case.

I hope this book is valuable and an enjoyable read compared to the average textbook and at the same time gives information and up-to-date knowledge for the care of critically ill patients. If I can save a single life with my education points, the purpose of this book will be fulfilled.

Badar M Zaheer

Acknowledgments

Saving a life is like saving a nation, as commanded by our faith, and I am thankful to our Creator for giving me this opportunity to serve his creations.

First of all, I would like to thank my family for their support and encouragement throughout the process. My wife Qudsia Zaheer MD and our children Fatima, Zubair, Naima, and Zaki Zaheer as well as my mother Nurjehan, for her prayers and blessings.

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Special thanks to Mr Suresh Kumar for his initial guidance in starting this book and Amer Aldeen MD, Professor of Emergency Medicine at Northwestern University, Sh. Zakaria Khudeira, Azhar Quader MD, Ayesha Sultana MD from CCN Chicago, and Abdel Azim El-Siddiq.

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Contents

CHAPTER

1

Head and Neck

Case Study 1: Subdural Hematoma

"I would especially like to commend the physician who, in acute diseases, by which the bulk of mankind are cutoff, conducts the treatment better than others"

—Hipporcrates

CASE HISTORY

A 55-year-old male presents to a small rural emergency department (ED) after a fall. The patient was attempting to fish during a beer festival event by standing on a rock. The patient slipped on the rock and subsequently fell and struck his head. On his arrival to the ED, his Glasgow coma scale (GCS) is 13. His alcohol level is 200 mg/dL. His complete blood count (CBC) is within normal limits and urine toxicology screen is negative. The Patient's vitals are unremarkable so it is deemed safe to obtain computed tomography (CT) scan of the head. A noncontrast CT scan reveals a subdural hematoma. A neurosurgeon at a nearby hospital is contacted and the patient is immediately airlifted to the awaiting operating room for surgery.

SUBDURAL HEMATOMA

Following Figures 1 and 2 represent the subdural hematoma and its treatment.

Discussion

Subdural hematoma is the most common cause of intracranial mass lesion.[1,2] It is defined as a collection of blood on the surface of the brain which may be acute, subacute or chronic. The acute type is usually caused by a high-speed impact to the skull. Risk factors include chronic alcoholism, epilepsy, coagulopathy, arachnoid cysts, anticoagulant therapy, cardiovascular disease, thrombocytopenia and diabetes. The cause is secondary to the shearing forces of small surface or bridging blood vessels. Subdural hematoma has a distinctive appearance on CT compared to epidural hematoma as it may appear as a crescent-shaped

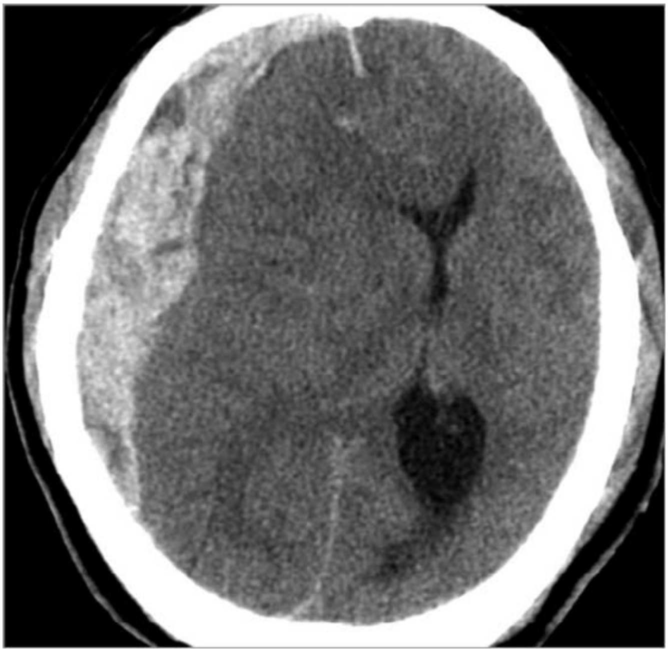


Fig. 1: Subdural hematoma
Source: http://www.ncbi.nlm.nih.gov/pmc/articles

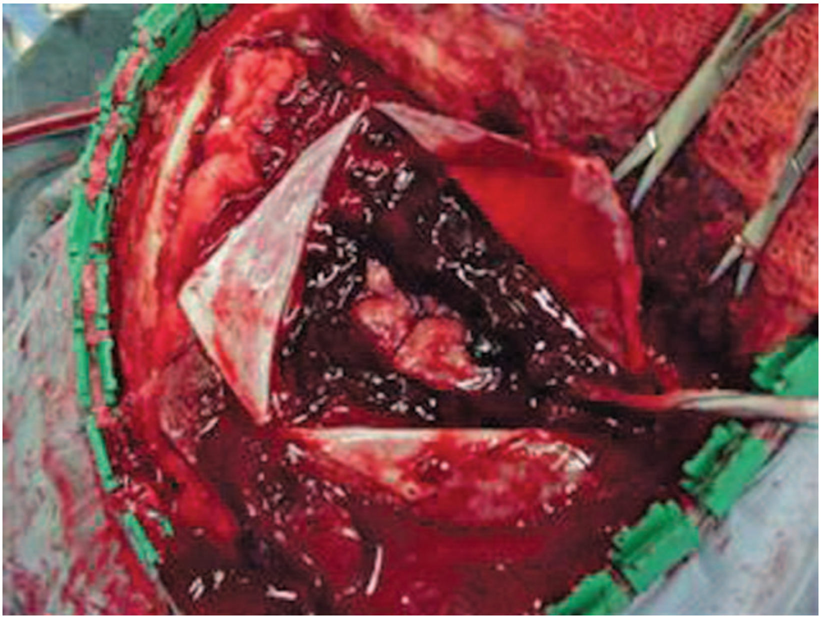
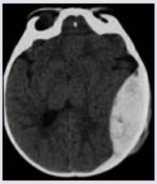
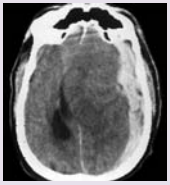
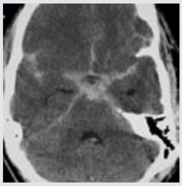


Fig. 2: Chronic subdural hematoma treatment
Source: Available online from http://www.health-reply.com/chronic-subdural-hematoma-treatment/

Table 1: Comparison between different intracranial hemorrhages

	Epidural hematoma	*Subdural hematoma*	*Subarachnoid hemorrhage*
Vessel involved	Arterial bleeding	Venous bleeding	Arterial-Venous
Presentation	Lucid interval	Progressively worsening headache	Thunderclap Headache, "worst headache of my life"
CT	Elliptical/lens shaped	Crescent	Star shaped, Texas "The Lone Star Flag"
Prognosis	Immediately fatal, if not evacuated	Slower progression than epidural. Fatal	Fatal if not treated
Risk factors	None	Elder persons, alcoholics, Alzheimers patients	Family history of Berry aneurysms
CT images (Figs 3, 4 and 5)	 **Fig. 3:** Epidural hematoma	 **Fig. 4:** Subdural hematoma	 **Fig. 5:** Subarachnoid hemorrhage

mass (Table 1). The risk of severe brain damage is much higher than with epidural hematoma and increased intracranial pressure (ICP) is correlated with a worse prognosis.

Presentation, which is usually gradual in onset, may include decreased level of consciousness, headache, difficulty in walking, cognitive dysfunction, personality changes, motor deficit and aphasia. It is essential to consult a neurosurgeon as soon as a subdural hematoma is suspected because the definitive treatment will require craniotomy.

Initial management should include the monitoring of airway, breathing and circulation (ABC), obtaining CT head, monitoring GCS, and obtaining laboratory tests values, such as that of CBC, coagulation profile, basal metabolic profile (BMP), type and screen, and drug and alcohol screening, to correlate clinical findings. Management should be done as discussed previously for decreasing ICP: start normal saline; elevate head of bed; optimized hyperventilation should be there and mannitol should be administered. Consider the procedure of creating burr holes in cases of rapid deterioration. Always maintain hemostasis when patients have been taking anticoagulants. Intubate if GCS is less than 12.

Modern term for trepanation is craniotomy which is used for epidural and subdural hematoma (Figs 6 and 7).

LEARNING POINTS FOR READING CT OF THE HEAD

Fig. 6: 18th century French illustration of trepanation.
Source: http://en.wikipedia.org/wiki/Trepanning

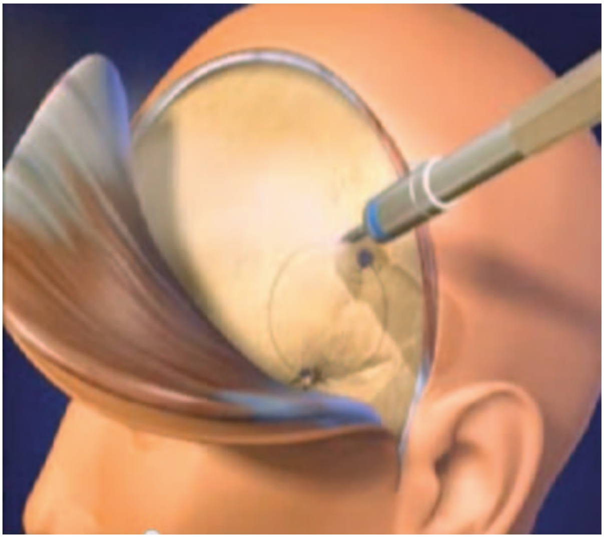
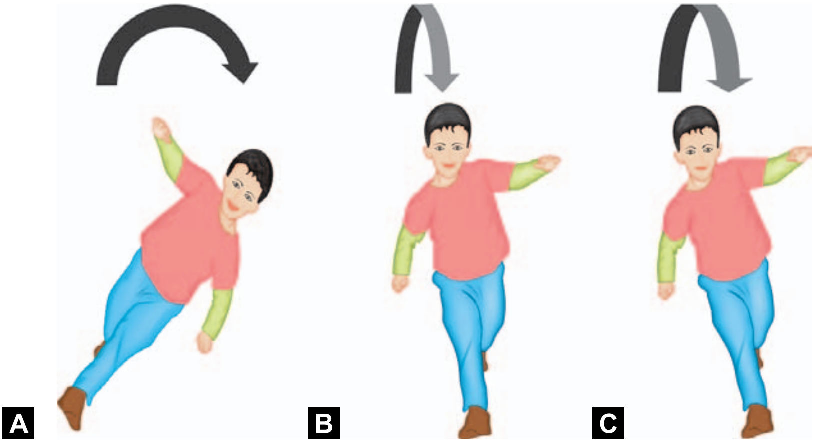


Fig. 7: Modern term for trepanation is craniotomy which is used for epidural and subdural hematoma.
Watch this video for a demonstration on Traditional Pterional Craniotomy by Hrayr Shahinian M.D.: http://www.youtube.com/watch?v=qfReIlmEEfU



Figs 8A to C: (A) Fall sideways highest risk; (B) Fall forward lowest risk and (C) Fall backwards moderate risk

Remember the mnemonic: A B B B C, when reading the CT Head
- *Air Sacs*: Sinuses; mastoid air cells fractures and infections
- Bones
- *Blood*: Different types of hemorrhages
- *Brain*: Infarction, edema, masses, and mid-line shift
- *CSF Spaces*: Ventricles, atrophy, hydrocephalus, edema

PRACTICE POINT

In children, if the child falls forward there is less risk of hemorrhage. However, if the child falls backwards it is moderate risk. Moreover, if the child falls on the side it is considered severe risk (Figs 8A to C).

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1. Subdural hematoma. Available online from www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001732/
2. Advanced trauma life support (ATLS). Subdural Hematoma. p. 139.

ADDITIONAL READING

1. http://www.youtube.com/watch?v=qfRellmEEfU

Case Study 2: Epidural Hematoma

"It is common to overlook what is nearby keeping the eye fixed on something remote."

—Samuel Johnson

CASE HISTORY

A 10-year-old school boy presents to the emergency department (ED) after being attacked by a gang. The boy reports that five individuals gathered around the boy and beat him with baseball bats. Upon initial presentation to the ED, the boy is neurologically intact with normal mentation and Glasgow coma scale (GCS) is 15. Initial evaluation includes unremarkable vital signs and some bruising throughout on examination. While finishing the remainder of the examination, the patient becomes stuporous and comatose. Repeat GCS has decreased to 7 and Pupils are now sluggish to react. Vitals are reassessed as temperature 37°C, (98.6°F) blood pressure 180/90 mm Hg, pulse rate 50 beats/minute, respiratory rate 12 breaths/minute and O_2 saturation level 93%. The patient is intubated and taken, immediately for computed tomography (CT) which reveals an epidural hematoma in the right temporal region. A neurosurgeon is called and the patient is taken immediately to the operation room for surgical evacuation of the bleed. What type of bleeding is this patient likely to have?

EPIDURAL HEMATOMA

Epidural hematoma is represented with the following Figures 1 and 2.

Epidural hematoma is defined as a collection of blood between the dura mater and the skull. It is most commonly caused by blow to the head. Generally the underlying brain tissue is sparse which makes the overall prognosis extremely good if an immediate action is taken.

Presentation

Only 20% of patients have the classic lucid interval. Patients may have severe headache, vomiting or seizures. Signs of increased intracranial pressure include hypertension, bradycardia and bradypnea. The medical professional should look for signs of herniation which would include a triad of coma, fixed pupils and decerebrate posturing in conjunction with contralateral hemiplegia. It occurs most commonly in the temporoparietal region due to location of the meningeal artery.

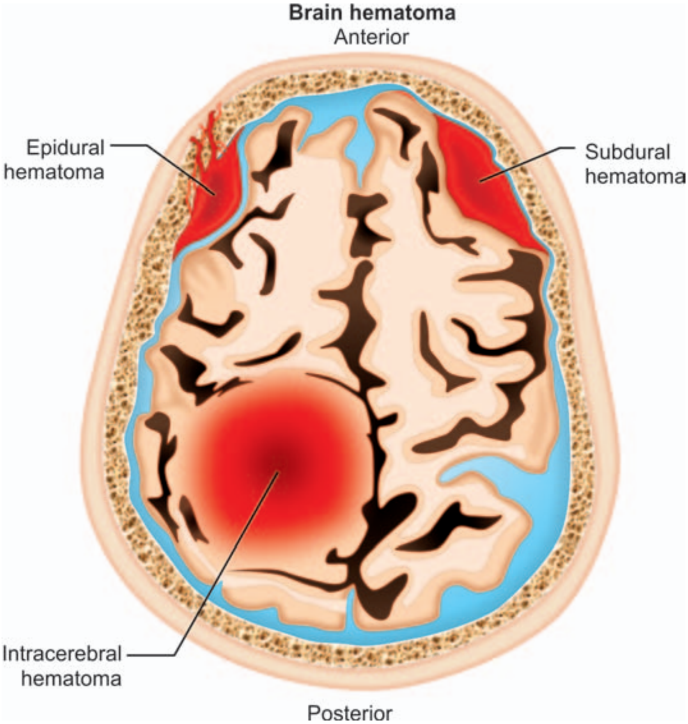

Fig. 1: Epidural hematoma

Source: Available online from http://www.images.emedicinehealth.com/images eMedicine Health/illustrations/brain-hematoma.jpg

Copyright: 2009, MedicineNet, Inc.

Fig. 2: Neurosurgical exposure of the epidural hematoma

Source: Singh P, Joseph B. Paraplegia in a patient with dengue. Neurol India [serial online]. 2010;58:962-3. Available from: http://www.neurologyindia.com/text.asp?

Management

Protect the airway by intubating; maintain blood pressure; elevate head of bed; consider optimized hyperventilation; Mannitol to reduce cerebral edema and phenytoin should be given for seizure prophylaxis. Early neurosurgical consult is an absolute requirement.

Conservative Method

A study of nonoperative management of epidural hematomas and subdural hematomas is done to investigate whether it is safe in lesions measuring one centimeter or less, (http://www.ncbi.nlm.nih.gov/pubmed/17693838) shows that EDH or SDH <1 cm thick can be safely managed nonoperatively unless there is concomitant cerebral edema.

Surgical Approach

Early involvement of a neurosurgeon is the most important duty of the emergency room MD. Surgical intervention is necessary for larger to intermediate epidural hematoma. It needs continuous careful individualized clinical approach based on radiological parameters: like thickness; midline shift; mass effect; and EDH location. For example, one of the study where a thickness of EDH > 18 mm, a midline shift > 4 mm, and a moderate to severe mass effect and the fourth criteria as location fairly predicted the outcome of epidural hematoma.

The surgical approach depends on the age, sex, GC score (Glasgow coma scale) mechanism of injury, interval between injury, and the CT scan findings.

Practice Point

A recent high-profile case of epidural hemorrhage occurred in 2009 when actress Natasha Richardson (Fig. 3) suffered a head injury while skiing. After the injury, Natasha experienced a lucid interval and dismissed the paramedics and ambulance assistance. She twice refused medical care, but three hours later she developed a headache and was taken to a local hospital. Despite the best efforts of physicians and transfer to two other hospitals, she died the following day. Autopsy revealed that she had suffered an epidural hematoma due to blunt impact to the head. This case has helped raise the public awareness of the dangers of head trauma and has provided a needed reminder that headache can be the first sign of a major pathology. During the lucid interval patients may be reluctant to agree to testing or hospitalization, emphasizing the importance of education on the dangers of epidural hematoma.

Fig. 3: Natasha Richardson: English actress of stage and screen who unfortunately died of epidural hemmorhage
Courtesy: Wikipedia

ADDITIONAL READING

1. http://www.ncbi.nlm.nih.gov/pubmed/17693838
2. http://www.ncbi.nlm.nih.gov/pubmed/9348150
3. Natasha Richardson (movie star): http://en.wikipedia.org/wiki/Natasha_Richardson

Case Study 3: Subarachnoid Hemorrhage

"If you can not make a judgment or a decision in 30 seconds, do not become an ER doctor"

—Badar M Zaheer

CASE HISTORY

A 30-year-old male presents to the emergency room (ER) and mentions to have experienced the worst headache of his life. The patient was sitting down for dinner after work, when all of a sudden he had severe pain in his head. The pain has been persistent for the last hour despite taking over-the-counter pain medications at home. The patient does not have a history of headaches in the past. He has faced no other medical problems in the past. He recalls that one of his relatives died suddenly at a young age from a bleed in his brain. The patient has smoked about a pack of cigarettes a day for the last 15 years. Vitals are as follows; temperature 38.5°C, blood pressure 190/100 mm Hg, pulse rate 120 beats/minute, respiratory rate 25 breaths/minute and O_2 saturation level 93% on room air. On physical examination, patient's alertness is waxing and waning. Emergency computed tomography (CT) has revealed subarachnoid hemorrhage (SAH). Nimodipine is started to control blood pressure. The head of bed is elevated to decrease intracranial pressure (ICP). Emergent neurosurgical consultation is obtained for evacuation of the bleed.

DISCUSSION

By definition, SAH is bleeding into the subarachnoid space which is the space between the brain and the tissues that cover it.[1] Causes include: arteriovenous malformation, bleeding disorders, cerebral aneurysms, head injuries, idiopathic and use of blood thinners. SAH from injuries is more common in the elderly population. In younger individuals, the most common cause is motor vehicle accidents. Bleeding from aneurysmal rupture happens in about 40–50/100,000 people over the age of 30 years. Risk factors include aneurysm in other blood vessels, fibromuscular dysplasia and other connective tissue disorders, high blood pressure, history of polycystic kidney disease and smoking. Family history of aneurysms also increases your risk.[2]

Clinically, the main symptom is a severe, sudden onset of headache near the back of the head. The pain may have started with a "popping" or "snapping" feeling also described as "thunderclap headache". Other symptoms include altered mental status, photophobia, mood and

personality changes, myalgia especially in the neck and shoulders, nausea and vomiting, loss of sensation, seizures, neck stiffness, vision problems including double vision, blind spots, vision loss, eyelid drooping and pupil size difference. Physical findings may include a stiff neck, focal neurologic deficits and/or decreased eye movements. Testing may include lumbar puncture, cerebral angiography, CT head, transcranial Doppler ultrasounds, magnetic resonance imaging (MRI) and magnetic resonance angiogram (MRA).

Goals of treatment are to repair the cause of bleeding, relieve symptoms, and prevent complications such as brain damage and seizures. Consultation from neurosurgeon should be done. Interventions may include a craniotomy and aneurysm clipping to remove pressure on the brain and close the aneurysm. Endovascular coiling may be used to reduce the risk of the aneurysm bleeding any further. In the absence of aneurysm, treat the increased ICP and monitor airway, breathing and circulation. Blood pressure should be controlled. Maintain systolic blood pressure to less than 160 mm Hg or maintain mean arterial pressure (MAP) of 110 mm Hg. Use nimodipine to prevent vasospasm in all patients.

Prognosis depends on the location and severity of the bleeding. The more complications, the worse the prognosis. Older age is also a poor indicator of outcome. Complications include repeated bleeding, coma or death. Other complications may occur as a result of the surgery, medication, seizures or stroke. New guidelines have been developed by the University of Ottawa for managing patients with suspected SAH.

The study applies to patients with nontraumatic headache and suggests them that they need further workup. The patient groups consist of:

- Age over 40 years, neck pain or stiffness, witnessed loss of consciousness, and onset occurs on exertion.
- Arrival by emergency medical services, age over 45 years, vomiting at least once, diastolic blood pressure higher than 100 mm Hg.
- Arrival by emergency medical services, age from 45 years to 55 years, neck pain or stiffness, systolic blood pressure higher than 160 mm Hg.

Although these guidelines are promising in identifying patients at the highest risk for SAH, results must be validated in other setting before being put into widespread use.

LESSON TO LEARN FROM A LEGAL POINT

A patient may present to the emergency department with a headache and may be treated as sinusitis, especially where there are residency-teaching programs. Unless we have a high index of suspicion for headaches, we may miss a subarachnoid hemorrhage, CNS tumors, etc. Diagnosis should

not be done hastily specially when you suspect a subarachnoid bleed or a tumor and you treat them with antibiotics for sinusitis or NSAIDS for headaches, the results may turn fatal. If a physician is not thorough in testing for SAH and the outcome results in death there will be hefty medicolegal and ethical consequences!

Figures 1 to 4 represent the subarachnoid hemorrhage.

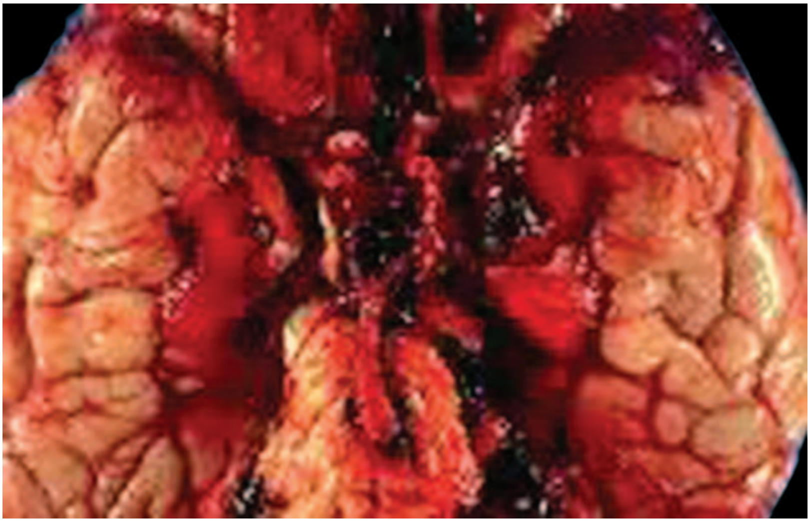

Fig. 1: Subarachnoid Hemorrhage
Source: http://emedicine.medscape.com/article/252142-overview

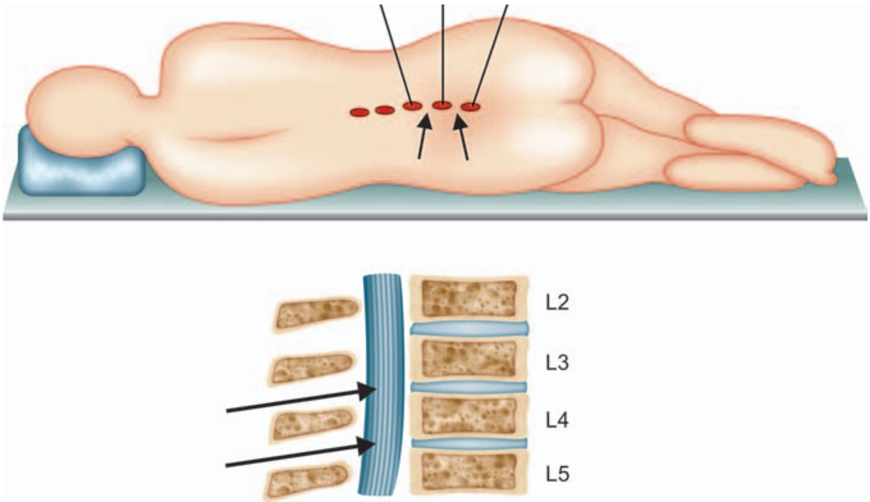

Fig. 2: Lumbar puncture locations
Source: http://www.cdc.gov/meningococcal/about/diagnosis-treatment.html

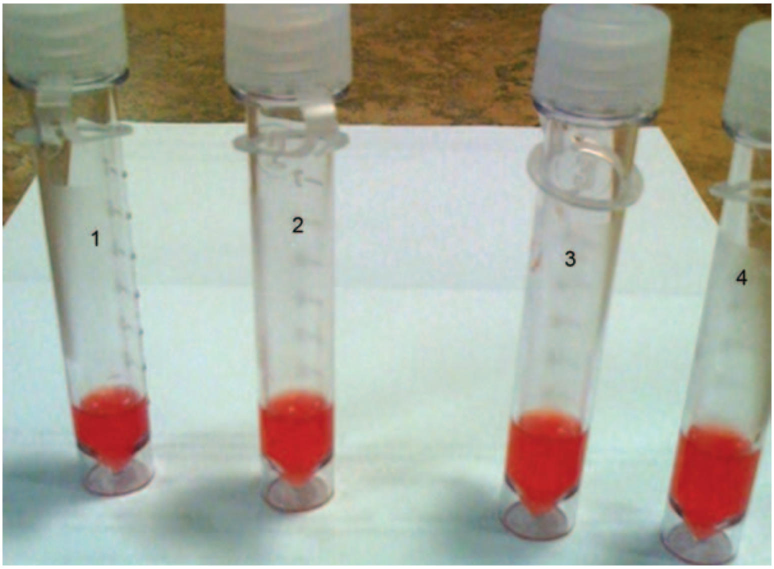

Fig. 3: Draw test tubes with cerebrospinal fluid (CSF) to evaluate for xanthochromia
Source: From Medscape

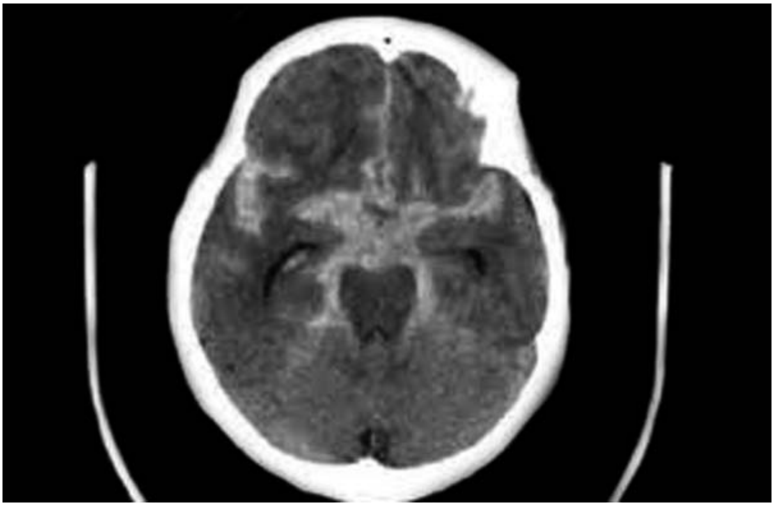

Fig. 4: Star Shaped, "The Lone Star Flag" Blood (bright white) is seen in the subarachnoid space on noncontract CT
Source: Badar M Zaheer

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2. Perry JJ, Stiel IG, Sivilotti ML, et al. High risk clinical characteristics for subarachnoid haemorrhage in patients with acute headache: prospective cohort study. BMJ. 2010;341:c5204. http://www.medscape.com/viewarticle/761589_2.

Case Study 4: Traumatic Brain Injury

"The wind doesn't always blow the direction you desire"

—Arab Proverb

CASE HISTORY

A 40-year-old female presents to the trauma bay after a motor vehicle accident (MVA). She was an unrestrained passenger in an accident in which a driver ran a red light and struck the driver side of her car. The driver also lost consciousness but was taken to another hospital. Initial assessment includes an intact airway with cervical collar in place. Patient is breathing spontaneously with adequate saturations. No signs of obvious bleeding are present. Distal pulses are all palpable and within the normal limits. Ecchymosis is found to be present periorbitally and on the abdomen. Glasgow coma scale (GCS) is 7. Due to GCS score, the patient is intubated before being assessed further. The patient is unresponsive and no family member is present. Vital signs are as follows: temperature 36°C, blood pressure 90/60 mm Hg and pulse rate 125 beats/minute. The patient is now on the ventilator. Normal saline bolus is given. Intracranial and intra-abdominal bleeding is suspected. Patient is sent for further imaging of head, cervical spine, chest and abdomen. Basilar skull fracture, subarachnoid hemorrhage and intra-abdominal hemorrhage due to liver laceration are revealed. Surgical and neurosurgical consultations are obtained. Careful monitoring of vitals is continued before the patient is sent to the operation room (OR) for definitive management.

DISCUSSION

Traumatic brain injury (TBI)[1] is a dynamic injury process due to cerebral edema, an increase in intracranial pressure (ICP) and anoxia (Fig. 1). It is usually related to rapid deceleration as seen in a motor vehicle accident, diving accident or blunt trauma. Initially, bleeding may be present followed by secondary injury due to cerebral edema. These injuries may have permanent consequences. There are 1.4 million cases in the United States per year with 50,000 deaths. A total of 235,000 hospitalizations and 1.1 million treated in the emergency department (ED). Of those that present to the ED, 80% are discharged, 10% are mild, and 10% are serious.

Causes include falls (28%), automobile accidents (20%), being struck by car (19%) and assaults (11%). Mild injuries are usually associated with a concussion because the effects are not generally life threatening. (Please see concussions case for further discussion).

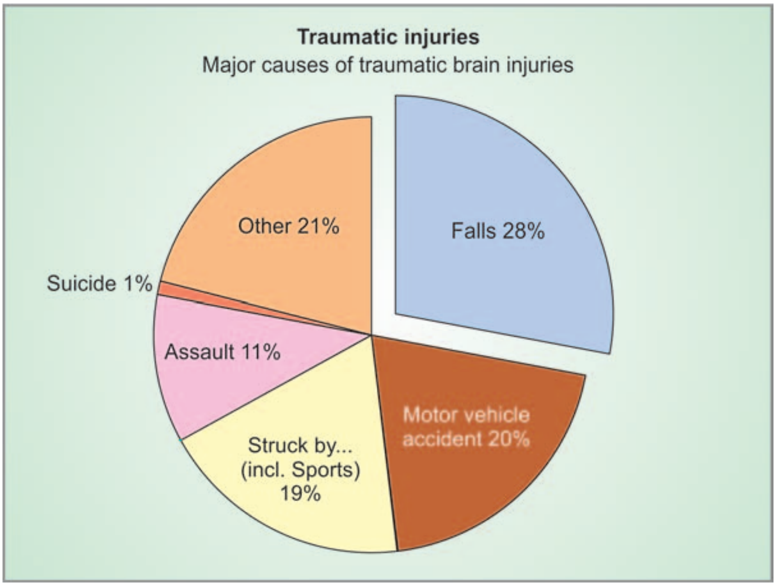

Fig. 1: Major causes of traumatic brain injuries
Source: National Center for Injury Prevention and Control, CDC

Severe TBI may lead to death or severe disability. There are two types of these injuries: closed and penetrating. Closed injuries are due to movement of the brain within the skull. These injuries result from falls, MVAs or blunt trauma. Penetrating injuries are caused by the entry of a foreign object into the skull. Initially assessment should include the GCS. Scores of 3–8 are consistent with severe TBI. Scores of 9–12 are associated with moderate TBI and finally scores of 13–15 are considered mild TBI.

Potential outcomes of severe TBI include coma, amnesia, decreased attention and memory, extreme weakness, impaired coordination and balance, loss of sensation including hearing, vision and perception loss, depression, anxiety, aggression, impulse control and personality changes. The effects of these symptoms impact both the patient's family and society.

Immediate treatment should be focused on maintaining airway, breathing and circulation (ABCs) and assessment using trauma protocols to assess for other injuries. All patients should receive 100% oxygen and two large bore intravenous (IV) lines. In severe cases, appropriate neurosurgery or neurology consultations should be obtained. Severely injured patients should be intubated and admitted to the appropriate intensive care unit (ICU) for further management. Goals should include controlling blood pressure, decreasing ICP and seizure prophylaxis. Cervical spine X-rays should be obtained due to the high-risk of associated cervical spine fracture. Emergent head computed tomography (CT)

should be obtained for all patients with GCS less than 14.[2] If subarachnoid hemorrhage is present, nimodipine should be administered to prevent vasospasm. [Please see subarachnoid hemorrhage (SAH) for further discussion]. A recent study suggests that measurement of plasma S100-B on admission of patients who have a minor head injury can be helpful to the physician to prevent ordering an unnecessary CT scan in certain low-risk cases.[3] The study shows that elevated S100-B proteins in the blood can be indicative of serious brain injury in patients.

PREVENTION

Educate your patients and community at large by supporting government laws and policies to prevent motor vehicle crashes and other sport related injuries. Educational handouts like a take-home point from the doctor are always helpful. Always wear your seat belt when traveling in the car and wear your helmet when traveling by motorcycle! Wear protective gear while playing contact sports.

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Case Study 5: Cervical Spine Injury

"Things cannot always go your way"

—Sir William Osler

CASE HISTORY

A 45-year-old African-American male lost control of his vehicle during a snowstorm. The vehicle flipped three times in a row before coming to rest in the ditch at the side of the road. The patient was extricated from his vehicle and transported to a nearby hospital. On arrival, the patient is immobilized and has a large scalp hematoma. All imaging findings, including cervical-spine (C-spine) X-ray, are interpreted as normal despite having a poorly visualized cervicothoracic junction (Figs 1 and 2). Based on negative studies and absence of pain, the nurse has removed the C-spine collar. After walking just down the hallway, the patient collapses. It is now found that the patient has experienced an anterior subluxation of the C-spine. Is this case approached correctly? How can we prevent spinal cord injury? What imaging is required to rule out C-spine injury during trauma?

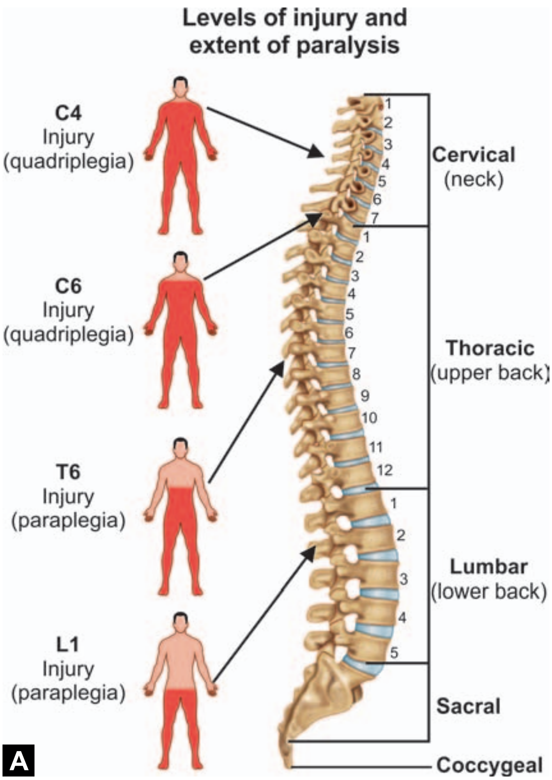
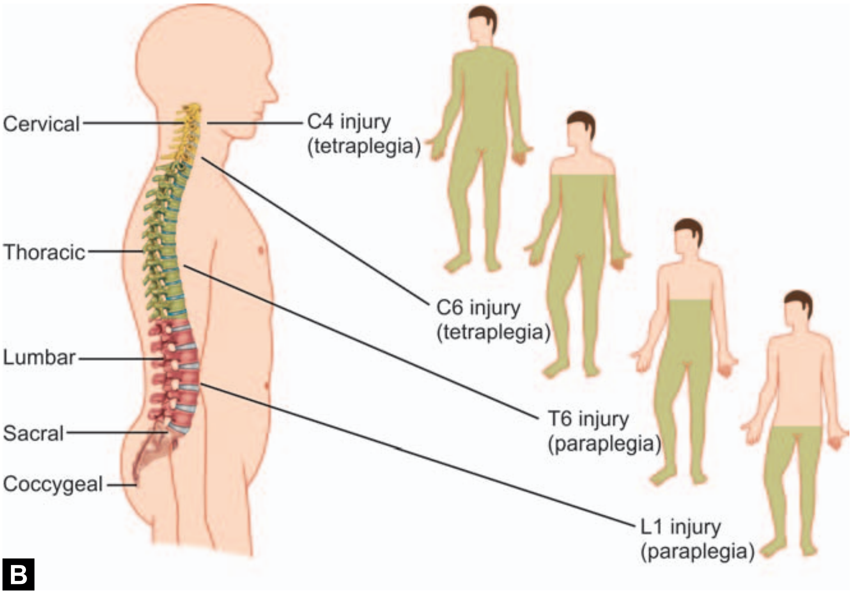


Fig. 1A



Figs 1A and B: Levels of injury and extent of paralysis
Copyright: Healthwise, incorporated

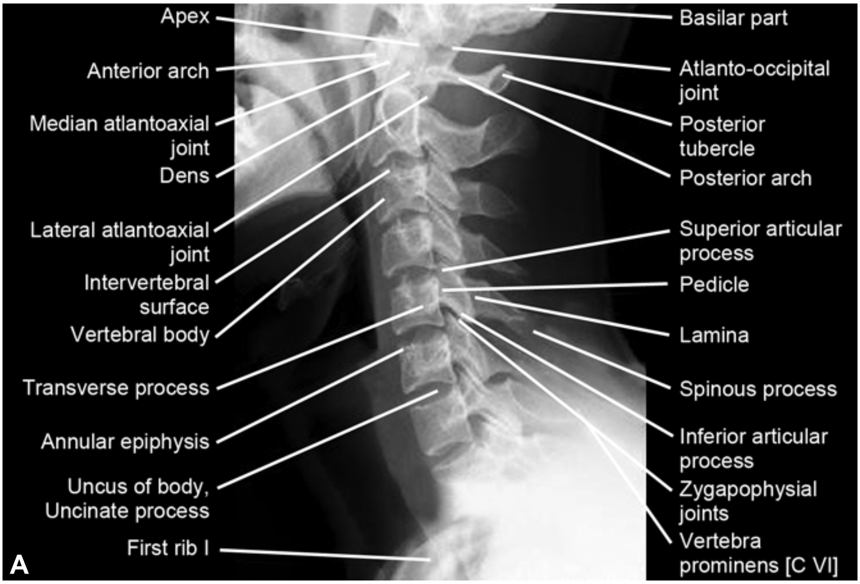


Fig. 2A

Figs 2A and B: (A) Anatomy of Cervical Spine; (B) C7 Fracture from (http://www.learningradiology.com/archives06/cow%20194-Brust%20fx/burstfxcorrect.htm)
Copyright: IMAIOS (for Fig. 2A)

	Radiography	CT
Cost	+	+ + +
Radiation	+	+ +
Time	+ + +	+
Sensitivity	94%	99%
Specificity	78–89%	93%
Technical Issues	+ + +	+

DISCUSSION

Cervical spine is the most vulnerable to injury because of its high mobility and exposure. The cervical canal is wider in the upper part from the foramen magnum to the lowest part of C2. The majority of patients with injuries around C2 vertebra who survive are neurologically intact when they arrive to the hospital. One-third of patients who have upper cervical injuries die at the scene of the injury secondary to apnea caused by an injury at C1 vertebra which denervates the phrenic nerves.

It is important to have a systematic approach for cervical spine assessment as not to miss any abnormalities. First, inspect for signs of blunt and penetrating injury, tracheal deviation and use of accessory respiratory muscles. Always palpate for tenderness, deformity, swelling, subcutaneous emphysema, tracheal deviation and symmetry of pulses. Obtain a computed tomography (CT) of the cervical spine or lateral, cross-table cervical spine X-ray. Throughout all evaluations and testing, always maintain adequate in-line immobilization and protection of the cervical spine.[2]

Certain guidelines are in place for when a C-spine collar may be removed. If a patient is awake, alert, sober neurologically intact, and without neck pain or midline tenderness then he is unlikely to have a C-spine fracture. Movement is generally safe when performed by the patient. However, if pain or midline tenderness is present, one must exclude a C-spine injury.[1]

As always, use a systematic approach when assessing bony films. Look for signs of bone deformity. Assess for fracture of the vertebral body or process. Look for loss of alignment of the posterior aspect of the vertebral bodies. Check for increased distance between the spinous process at one level. Assess for narrowing of the vertebral canal and increased prevertebral soft tissue space.

Recent developments in treatment of cervical spine injuries include the use of stem cells to help regrow nervous tissue in the cervical spine.

Ten percent of patients with cervical spine injury will have a second noncontiguous vertebral fracture. If the spine is protected, further examination can be deferred until ABCDs are addressed. Look for the presence of hypotension, especially bleeding in other organs. Also, look for the causes of respiratory inadequacy.

PRACTICE PEARL

- CT is cost effective if fracture risk > 4%
- CT saves money if fracture risk > 10%

Always remember the cost of law suit by missing one fracture which changes the life of the person. Our job is not to hurt the patient but at the same time you do not want to get hurt yourself by getting involved in a law suit.

Pitfall

Don't leave the patient on a hard surface, such as a backboard, for a long period of time. This may lead to formation of serious decubitus ulcers in patients with spinal cord injuries. The patient should be evaluated by the appropriate specialist and removed from the spine board as quickly as possible. Nobody should be left on the spine board for more than 2 hours. If a patient has to be immobilized for more than 2 hours, they must be logrolled every 2 hours, maintaining the integrity of the skin and spine.

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1. http://www.youtube.com/watch?v=s7gULAPLY8U
2. American College of Surgeons Committee on Trauma. (ATLS) Advanced Trauma Life Support for Doctors: Student Course Manual, 8th edition.

Case Study 6: Atypical Headache

"Gathering the appropriate studies in advance saves a lot of headaches."

—Badar M Zaheer

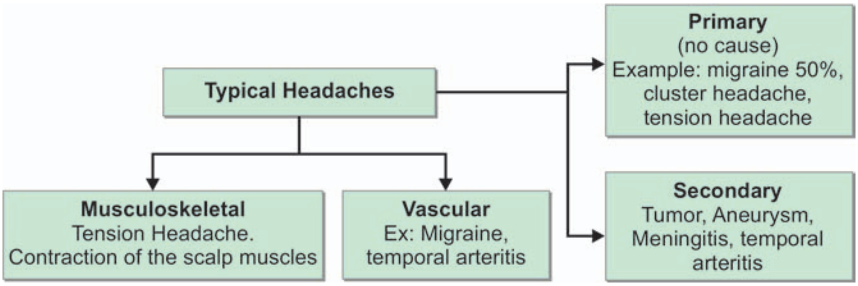
CASE HISTORY

A 40-year-old female presents to the emergency department (ED) with a headache persisting over the past several weeks. The patient has never had headaches like this before. The headache is unilateral on the right side. The patient is thought to have migraine headaches and is discharged home with oral analgesics for pain and antiemetics to control her nausea. One week later, emergency medical service (EMS) is called again because the patient is found to be unresponsive. Narcan is given which results in some mild improvement. The patient is thought to have been overusing hydrocodone and is sent home with further instructions for treating her migraine headache. The patient is dissatisfied with this diagnosis because she has never had migraine headaches before in her life. Subsequently, she decides to go to a different hospital. This time, computed tomography (CT) head is ordered and the patient is found to have a large right frontal tumor. Before surgery can be scheduled, the patient passes away.

DISCUSSION

This sad case can be taken as a lesson to look for red flag signs in headaches. Causes of headaches can range from simple tension headache, to subarachnoid hemorrhage, to malignancy and therefore must be taken seriously. In this case, the patient warranted a CT scan due to the new onset of the headaches. It would also be important to assess her neurologic status, because a tumor of this size may have caused neurologic abnormalities. Neurologic assessment may have been missed because the patient was incorrectly assessed as having abused her

Flow chart 1: Types of Headache



Source: Badar M Zaheer

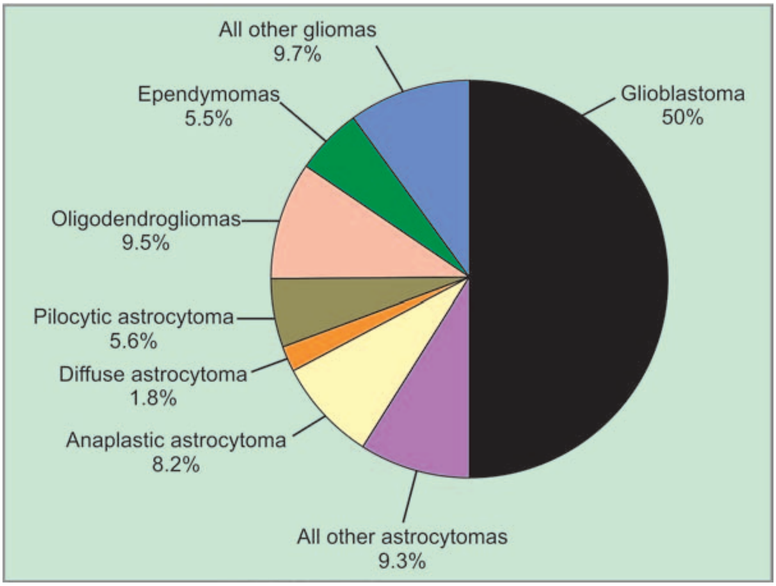


Fig. 1: Frequency of brain astrocytomas
Source: Neurosurg focus copyright 2006 American Association of Neurological surgeons

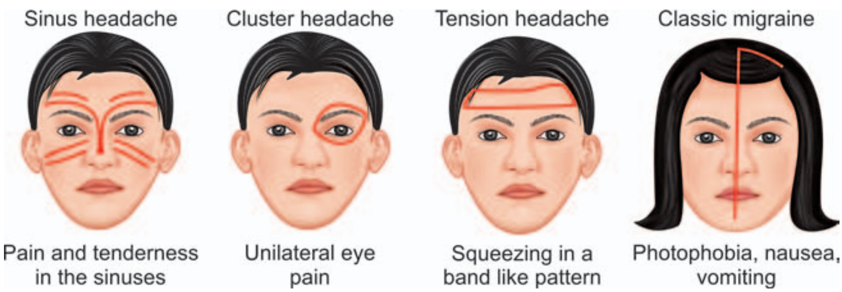


Fig. 2: Pain patterns in different types of headaches
Source: Badar M Zaheer

narcotic medication. This case also stresses the need for close monitoring and follow-up to ensure that patients do improve once they leave the ED.

Glioblastomas (malignant glioma) are the most common primary adult malignant brain tumors, (Fig. 3) and 20% of all primary brain neoplasms are glioblastoma multiforme (GBM) tumors.[1] Glioblastoma multiforme is the highest-grade form of astrocytoma and makes up about two-thirds of all brain astrocytomas (Fig. 1). The prognosis for this tumor is at the extreme worst end because of its high-grade status. Overall, metastatic tumors are the most common cause of brain neoplasms (Fig. 2).

Most of the metastatic tumors are caused by cell mutations. A history of irradiation to the head may also increase the risk of brain tumor development. In some inherited diseases may be the cause and should be

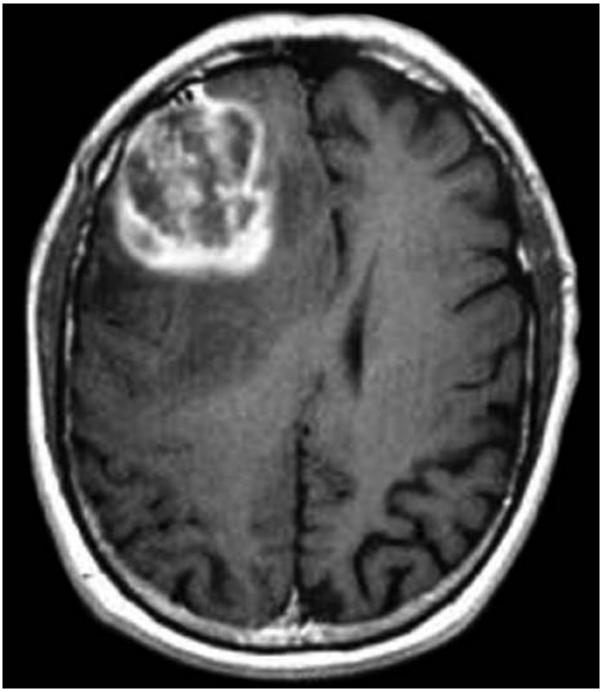


Fig. 3: Most common brain tumor, Glioblastoma (Stage IV astrocytoma)
Source: http://emedicine.medscape.com/article/340870-overview

checked for with a patient and familial history. In HIV patients, primary central nervous system (CNS) lymphoma is a frequent cause of neoplasm. For metastatic neoplasms, lung is the most common cause of metastasis.

Presentation may include symptoms of headache, (see Flow chart 1) altered mental status, ataxia, nausea, vomiting, weakness and gait disturbance. More focal symptoms include seizures, visual changes, speech deficits or and focal sensory normalities. Symptoms usually have a gradual onset. Complaints of headaches, one of the least-tolerated symptoms, occur later on and usually do not present alone. The headaches usually seem like tension-type nonspecific headaches. Mental status changes may be seen depending on the area of brain that is affected. Contrary to headaches, seizures may be an early sign, and are usually focal or generalized.

In general, ED management is not dependent on the type of tumor. The main concerns in the ED is managing intracranial pressure (ICP) that may result from the edema caused by the tumor. As a result of increased ICP, cerebral circulation may be impaired as well as shifting and herniation.

Physical examination should include a complete neurologic assessment. Look for localized deficits, papilledema, diplopia, impaired upward gaze, visual field deficits, anosmia, cranial nerve palsies, ataxia,

nystagmus or sensory deficits. When a neoplasm is suspected, patients should be screened with basic laboratory tests because they are at higher risk for medical complications, bleeding, and metabolic and endocrine disorders. CT is usually the first type of imaging done in the ED due to the ease of obtaining this test. Intravenous contrast CT can be used. Magnetic resonance imaging (MRI) is the preferred choice of imaging and should be done initially, if possible, but will be required either way for further management. Airway, breathing and circulation (ABCs) should always be addressed. Cerebral edema may be treated with corticosteroids. Dexamethasone 4–24 mg daily may be used. Patients are generally admitted for further workup and appropriate consultations should be obtained including neurosurgery.

LESSONS FROM THE COURT

Keep in mind, every headache can be a big headache.

Key pitfalls to avoid malpractice lawsuits:

- Failure to diagnose by taking proper history and appropriate physical exam (42%).
- Failure to refer to a proper specialist.
- Failure to follow-up.
- Failure to order a proper diagnostic test (55%).

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Case Study 7: Concussion

Always wear your seat belt and remove tripping hazards in the home. Prevention and appropriate response to TBI can help save lives—CDC

—Badar M Zaheer

CASE HISTORY

A 12-year-old male presents to the emergency department (ED) after being knocked out during a football game (Fig. 1). The patient was running with the football when he was tackled, the opposing player put his helmet into the patient's helmet causing his head to fly back violently and he lost consciousness for a few seconds. The patient is experiencing some nausea and has had several episodes of vomiting. He has never had a concussion before, other than the brief episode of loss of consciousness, he denies amnesia of the event. On examination, vitals are temperature 36.8°C, blood pressure 108/70 mm Hg, pulse rate 65 beats/minute, respiratory rate 15 breaths/minute, and O_2 saturation level 100% on room air. The patient is found to be neurologically intact on cranial nerve, strength, sensation and reflex examination. He is given Zofran and Tylenol and is then observed in the ED. Because the patient begins to experience worsening nausea a CT head is done which is found to be unremarkable. What is the next best step in management? What needs to be done before the patient can return to play?

DISCUSSION

A concussion is a complex process affecting brain function induced by a traumatic biochemical force. Concussions predominantly occur between the ages of 12 years and 24 years, and more commonly in males. Risk factors include contact sports (especially football) and recent concussion (Figs 2A to C). Prevention should include educating coaches and athletes, rule enforcement and possibly rule changes. Current protective headgear has not been shown to prevent concussions however they are still mandatory. Improvement of such devices are being researched and developed.

Symptoms include confusion, post-traumatic amnesia, retrograde amnesia, loss of consciousness, disorientation, a "foggy feeling" inability to focus, delayed verbal and motor responses, slurred/incoherent speech, excessive drowsiness, headache, fatigue, disequilibrium, dizziness, visual disturbances, phonophobia, emotional liability, irritability and sleep disturbance. Severity can only be assessed retroactively.

Fig. 1: Traumatic Brain Injury
Source: Available online from http://www.cdc.gov/traumaticbraininjury/pdf/blue_book.pdf

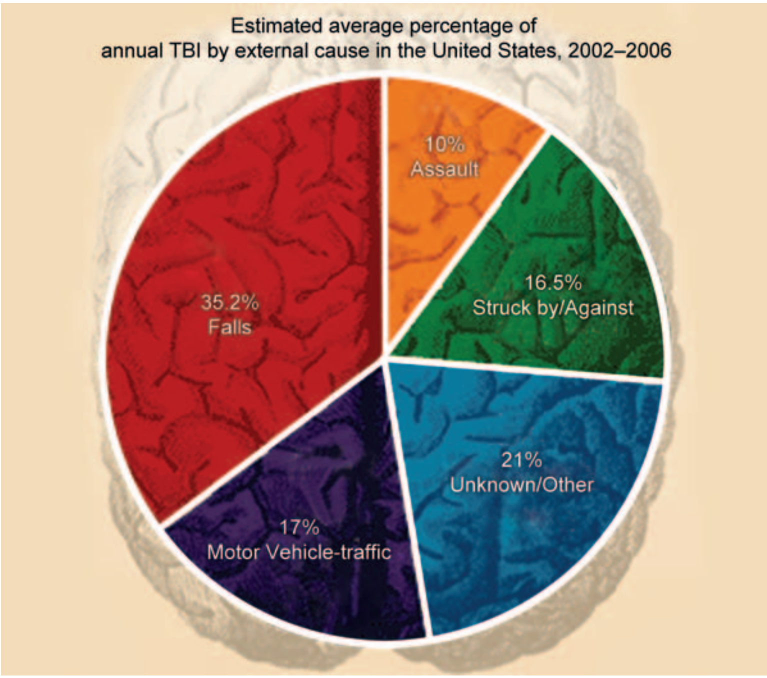

Fig. 2A: Estimated Average Percentage of Annual TBI
Source: Available online from http:// www.cdc.gov/features/dstbi_braininjury/

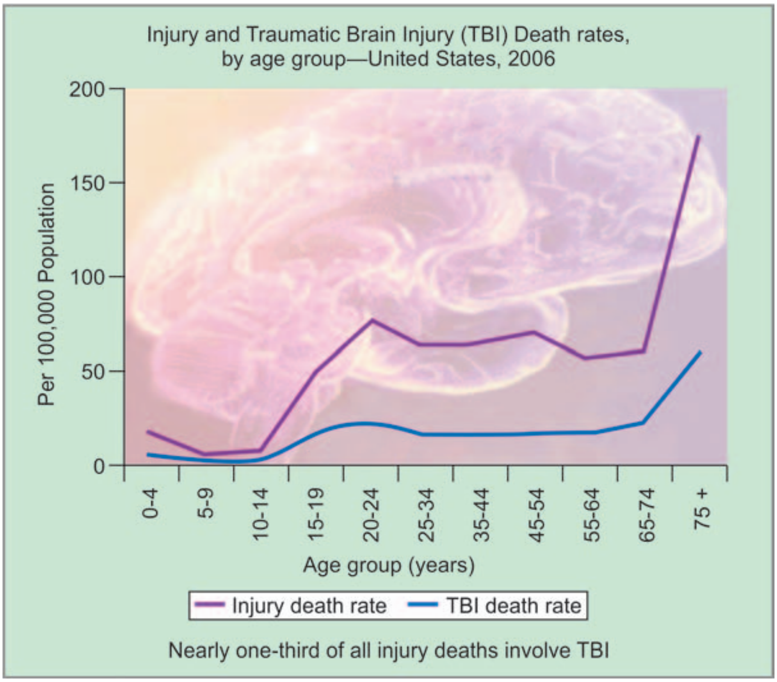

Fig. 2B: TBI Death Rates

Source: Available online from http://www.cdc.gov/features/dstbi_braininjury/

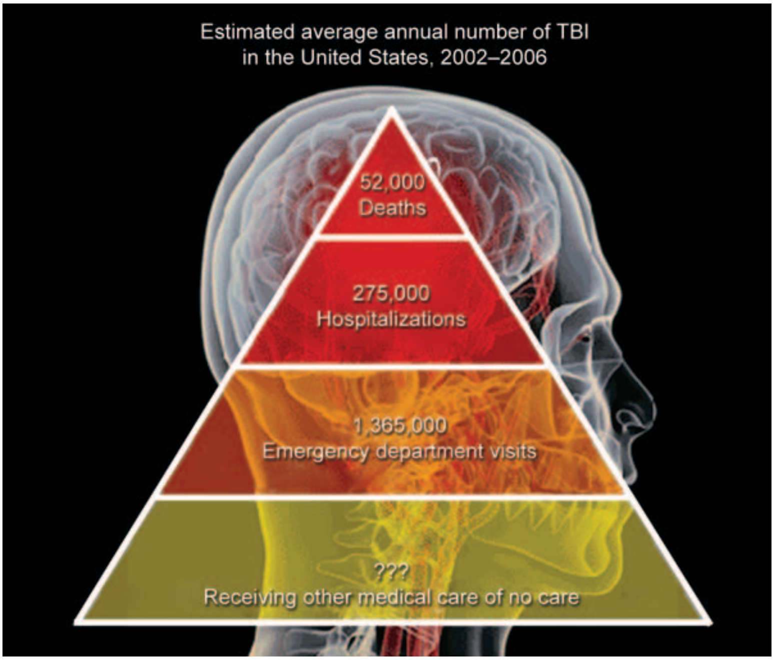

Fig. 2C: Estimated Average Annual Number of TBI

Source: Available online from http://www.cdc.gov/features/dstbi_braininjury/

PHYSICAL EXAMINATION

The physician should assess for airway, breathing and circulation (ABC), evidence of trauma, neurologic examination and cervical spine injury. Sideline assessment of concussion and serial cognitive evaluations may be used to determine if patient has returned to baseline. Patient must be assessed by a primary care doctor, not an emergency medicine physician in order to return to play.

Imaging is typically normal and should be considered if there is prolonged loss of consciousness, focal neurologic deficits or worsening of symptoms. Cervical spine films should be ordered as indicated. Differential diagnosis includes concussion, subdural hematoma, epidural hematoma, cerebral contusion, and facial or skull fracture.

TREATMENT

Treatment includes rest from physical and mental activity because both may worsen symptoms. i.e., the patient should remain in place until they are symptom free. Patients should re-initiate activity in a stepwise fashion with light activity first. Delay activity by 24 hours if concussive symptoms return and then repeat. Ibuprofen or acetaminophen may be used for pain management. Treatment can usually be done by a primary care physician. More complicated cases should be referred to a sports medicine specialist or neurologist. Education regarding postconcussion symptoms and their treatment is very important. Complications to look for include delayed hematomas and recurrent concussion syndrome. Chronic traumatic brain injury can lead to cognitive decline and parkinsonian type syndromes.

With more knowledge being gained about concussion management, physicians, coaches and players often disagree about playing time.[1] Coaches and players often want themselves to get right back into the game despite recommendations from the physician. More precautions regarding concussion management are being taken now more than ever.

Basic sideline management for concussions has been generally accepted, although randomized control data has been limited. If a patient shows any signs of a concussion, the player should be medically evaluated on the sideline and assessed for cervical spine injury. If no health care professional is present, then the player should be sent for further evaluation. The player should be initially treated with first aid guidelines and then assessed for signs and symptoms such as loss of consciousness, headache, amnesia, nausea or dizziness. Player should be

accompanied by at least one friend or family member for the next 5 hours. Players should not be allowed to return to play on the same day of injury with the exception of some adult athletes.[2]

VIENNA CONCUSSION CONFERENCE: RETURN TO PLAY RECOMMENDATIONS

Athletes should complete the following stepwise process prior to return to play following the concussion:

1. Removal from contest following any signs/symptoms of concussion.
2. No return to play in current game.
3. Medical evaluation following injury:
    a. Rule out more serious intracranial pathology.
    b. Neuropsychologic testing (considered a cornerstone of proper postinjury assessment).
4. *Stepwise return to play*:
    a. No activity and rest until asymptomatic
    b. Light aerobic exercise
    c. Sport-specific training
    d. Noncontact drills
    e. Full-contact drills
    f. Game play.

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1. O'Reilly KB. "Put me in, Doc." American Medical News: Professional Issues. 2010.
2. Stevenson JH, "Concussion." In: Domino FJ (Ed). The 5-minute clinical consult: 2012, 20th edition.

ADDITIONAL READING

1. http://www.cdc.gov/features/dstbi_braininjury/

CHAPTER

2

Chest

PULMONARY SYSTEM

Case Study 8: Pulmonary Hypertension

CASE HISTORY

Chief Complaints: Syncope and Dizziness

History of Past Illness

Mrs A is a 36-year-old female who is presented to the emergency department (ED) after an episode of syncope and continued dizziness. She was singing in church and suddenly felt dizzy and fell. She has been an avid church goer for much of her life and recently experienced the sudden passing of her aunt whom she considered a mother figure, she was 48 years of age and experienced a long battle with heart problems. The patient has been struggling with obesity for much of her life and has been taking "weight loss pills" in addition to starting multiple diet and exercise regiments.

On the way to the ED, the patient developed dyspnea and tachypnea. At the ED, she complains of chest pain, palpitation and an on-and-off dry cough.

She denies any previous fainting episodes. She denies any history of seizure.

PHYSICAL EXAMINATION

- *Cardiovascular*: Jugular venous pressure (JVP) is raised; Loud P2, S3 and S4 is heard.
- *Respiration*: Tachypnea, no wheezing is heard.
- *Abdomen*: On palpation, slight hepatomegaly is found.
- *Extremities*: Pedal edema +1.
- *Central nervous system (CNS)*: Intact.

DIAGNOSTIC TESTS

- *Electrocardiogram (ECG)*: Right ventricular hypertrophy and right axis deviation.
- *Pulmonary function test (PFT)*: Arterial hypoxemia, reduced diffusion capacity, hypocapnia.
- *Ventilation/Perfusion lung scan (V/Q scan)*: No proximal pulmonary artery emboli.

Laboratory Tests

- Antinuclear antibody (ANA) is positive
- D-Dimer is negative

Imaging

- *Chest X-ray (CXR)*: It shows enlarged central pulmonary arteries and right ventricular enlargement.
- *Echocardiography*: It shows right ventricular enlargement and overload. No atrial septal defect (ASD) is present and low mitral stenosis is found (see Fig. 4)

DIAGNOSIS: PULMONARY\ ARTERIAL HYPERTENSION

Differential Diagnosis (DDX): For Patient with Syncope and Dizziness

- *Cardiovascular*: Aortic stenosis, asymmetric septal hypertrophy, ventricular tachycardia, supraventricular tachycardia, sinus node disorders (bradycardia), atrioventricular (AV) block.
- *Hemodynamic*: Decreased total peripheral resistance (TPR), subclavian steal, stroke, orthostatic hypotension.
- *Respiratory*: Pulmonary embolism, pneumothorax, chronic obstructive pulmonary disease (COPD) exacerbation, asthma exacerbation.
- *Endocrine*: Hypoglycemia, Addison's disease (decreased TPR).
- *Neurologic*: Vasovagal reflex, complex seizure.
- *Neoplasm*: Rare, but needs to be considered.
- *Drugs*: B-blockers, vasodilators, nitrates, tricyclic antidepressant, phenothiazides, barbiturates, benzodiazepines, psychotropic drugs, alcohol.

WORK UP: PULMONARY HYPERTENSION

Rule Out Secondary Causes

Pulmonary hypertension can often be secondary to other conditions. Left heart failure, for example, increases the resistance of blood flow away from the lungs. Conditions like COPD or sleep apnea can increase pulmonary blood pressure by causing vasoconstriction in areas of the lung where there is inadequate ventilation. Secondary causes of pulmonary hypertension need to be excluded first (Table 1).

Table 1: World Health Organization's Diagnostic Classification of Pulmonary Hypertension

Pulmonary arterial hypertension	*Pulmonary hypertension associated with disorders of the respiratory system and/or hypoxemia*
Primary pulmonary hypertension	Chronic obstructive pulmonary disease
Sporadic disorder	Interstitial lung disease
Familial disorder	Sleep-disordered breathing
Related conditions	Alveolar hypoventilation disorders
Collagen vascular disease	Chronic exposure to high altitudes
Congenital systemic-to-pulmonary shunt	Neonatal lung disease
Portal hypertension	Alveolar-capillary dysplasia
Human immunodeficiency virus infection	Others
Drug and toxins	*Pulmonary hypertension resulting from chronic thrombotic and/or embloic disease*
Anorectic agents (appetite suppressants)	Thromboembolic obstruction of proximal pulmonary arteries
Others	Obstruction of distal pulmonary arteries
Persistent pulmonary hypertension of the newborn	Pulmonary embolism (thrombus, tumor, ova and/or parasites. foreign material)
Others	In-situ thrombosis
Pulmonary venous hypertension	Sickle cell disease
Left-sided atrial or ventricular heart disease	*Pulmonary hypertension resulting from disorders directly affecting the pulmonary vasculature*
Left-sided valvular heart disease	Inflammatory conditions
Extrinsic compression of central pulmonary veins	Schistosomiasis
Fibrosing mediastinitis	Sarcoidosis
Adenopathy and/or tumors	Others
Pulmonary veno-occlusive disease	Pulmonary capillary hemangiomatosis
Others	

Source: WHO[1]

Fig. 1: Chest X-ray shows enlarged central pulmonary arteries and right ventricular enlargement

Diagnostic Studies

Chest X-ray and High Resolution CT Scan

In pulmonary hypertension these studies show dilation and pruning of pulmonary arteries, enlargement of right atrium and right ventricle. These studies can help to rule out other more obvious lung pathology (Fig. 1).

Prominence of both hilum due to enlargement of pulmonary vessels. There is blunting of both costophrenic angles which could represent small pleural effusion/pleural reaction. There is pruning of peripheral pulmonary vessels; findings are consistent with pulmonary hypertension. Refer to Figure 4 for ultrasound findings in pulmonary hypertension.

Electrocardiography

This can show right atrial dilation, right bundle branch block (RBBB), right atrial enlargement and right ventricular hypertrophy (Fig. 2).

Criteria for right bundle branch block:

- QRS duration of greater than or equal to 120 milliseconds.
- rsR' "bunny ear" pattern in precordial leads.
- Slurred S waves in leads I, V5 and V6.

Source: http://www.learntheheart.com/RBBB.html

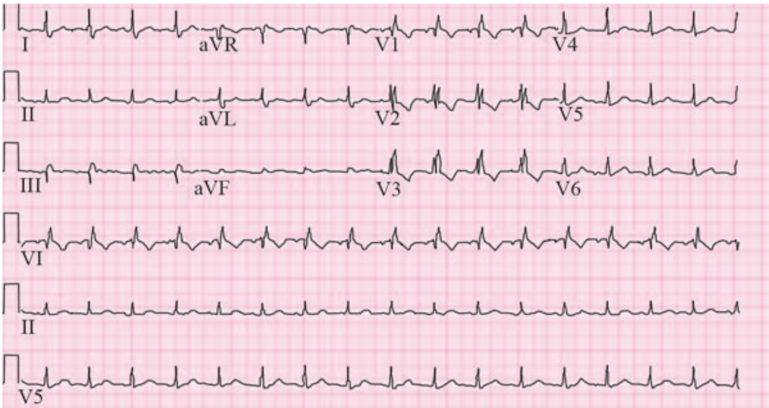


Fig. 2: ECG shows right bundle branch block (RBBB), right atrial enlargement and right ventricular hypertrophy

Pulmonary Function Test

- Decreased diffusion capacity of the lung for carbon monoxide (DLCO), only mild restrictive pattern [decrease in forced expiratory volume in 1 second (FEV1), rules out restrictive lung disease]

Arterial Blood Gases (ABG) and Polysomnography (Sleep Study)

- Show decreased arterial O_2 concentration and saturation
- Show and increased A—a gradient
- Used to rule out hypoventilation and obstructive sleep apnea (OSA).

Acute Vasodilatory Test

- It is performed during cardiac catheterization, and it is done to screen for responsiveness to calcium channel blocker.
- A positive acute vasodilator test response means better survival.
- It can be performed on all patients who have idiopathic pulmonary hypertension, and who are candidates for long-term calcium channel blocker (CCB). Medications used to perform this test include inhaled nitrous oxide, intravenous (IV) epoprostenol and IV adenosine.
- Cardiac catheterization is contraindicated in right heart failure and if the patient is hemodynamically unstable.

TREATMENT

The major treatment goals in pulmonary hypertension are the inhibition of vasoactive substances in lungs and prevention of right ventricular failure (see Fig. 3).

Oral Calcium Channel Blockers

Calcium channel blockers are a great choice for patients that have a positive acute vasoactive response. Half of all the patients will have a long-term response and a significant decrease in mortality. Side effects of CCB include hypotension and lower limb edema.

Intravenous Prostacyclin

These medications include epoprostenol and flolan. They act by vasodilation, decreasing platelet aggregation and decreasing smooth muscle proliferation. These benefits are thought to increase with time. They have been shown to also decrease mortality. Adverse effects include flushing, headaches, jaw or leg pain, abdominal cramps, nausea and diarrhea.

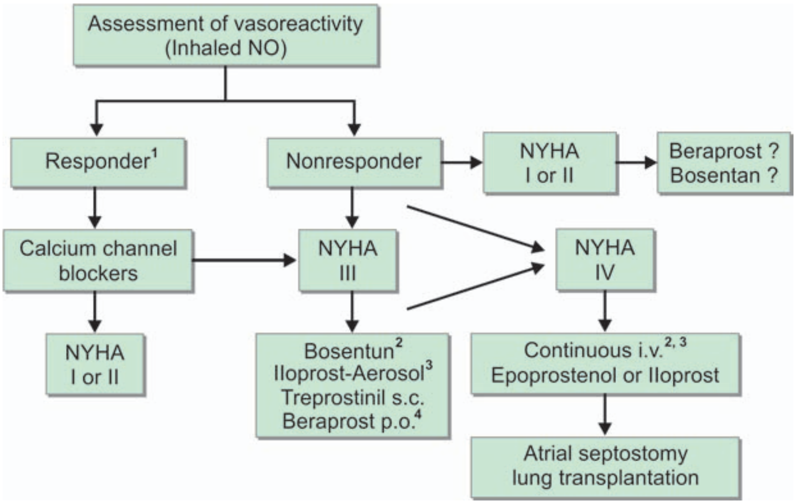


Fig. 3: Treatment of pulmonary arterial hypertension. Initial therapy should be guided by the results of acute vasodilator challenge. (1) In patients who show a fall in pulmonary arterial pressure and pulmonary vascular resistance to near-normal values, calcium channel blockers remain a reasonable therapeutic option. Nonresponders and responders who are not in NYHA Class I or II while being treated with calcium channel blockers should be offered the endothelin receptor antagonist bosentan or one of the novel prostaglandins. In case of deterioration or in patients with advanced disease, intravenous epoprostenol or iloprost should be started and atrial septostomy or lung transplantation should be considered. (2) The combination of epoprostenol and bosentan is currently being evaluated in clinical studies. (3) Iloprost is not available in the United States and has not received official approval for treatment of pulmonary hypertension in Europe. (4) Some of these substances have not been approved in the United States and Europe

Source: http://ajrccm.atsjournals.org/content/165/9/1209/F.larage.jpg

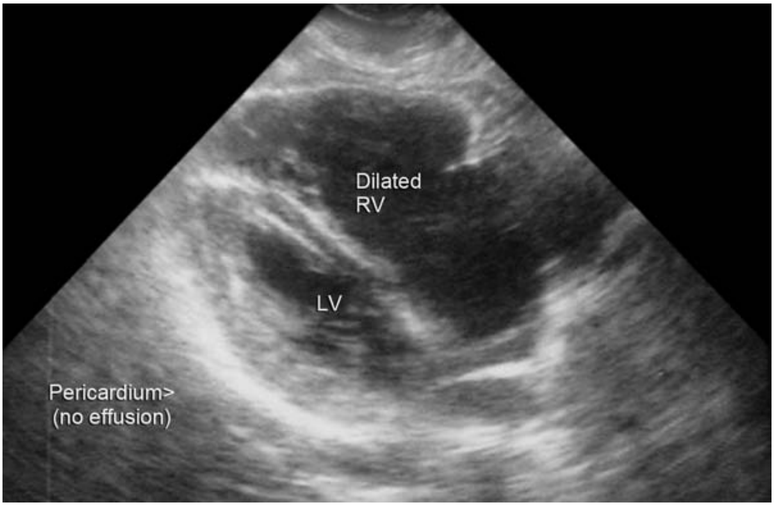


Fig. 4: Ultrasound shows dilated RV
Source: Badar M Zaheer MD

Prostacyclin Analogues

Iloprost (inhaled), Treprostinil (IV or subcutaneous), Beraprost (per oral) have the same mechanisms as the IV prostacyclins. They are easier to adhere to and have decreased side effects. Iloprost has the most increased efficacy.

Endothelin Receptor Antagonists

These agents include bosentan and ambrisentan. They cause a decrease in smooth muscle remodeling, increase in vasodilation, decrease in fibrosis, decrease in symptoms and increase in 6-minute walking test. However, it has no change in clinical outcomes. They have a low adverse-effect profile.

Pearls

- Removal of secondary causes is the mainstay treatment.
- CXR and ECG are a good starting point.
- Oral calcium channel blocker and IV prostacyclin—only medications proven to lower mortality.

REFERENCE

1. World Health Organization: http://www.who.int/respiratory/other/Pulmonary_hypertension/en/

ADDITIONAL READING

1. Quick Essentials Emergency Medicine 4.0, EMresource.org

Case Study 9: Pulmonary Embolism

"Every moment of your life is an opportunity to learn and perfect your skills to save a life."

—Badar M Zaheer

CASE HISTORY

The patient is a 61-year-old female refugee from Afghanistan with an unknown previous medical history. She is presented to an urgent care clinic with a chief complaint of vague abdominal pain and a long history of back pain that radiates to the back of the leg. She was seen in emergency room 1 day prior, with similar symptoms. Abdominal and pelvic X-rays are done and they show no significant pathology. Doppler ultrasounds are done in both legs and they show negative findings for deep venous thrombosis (DVTs). The patient is given morphine and zofran and then sent to home.

Soon after she says that her symptoms have not improved. The patient is given a gastrointestinal (GI) cocktail which consists of three ingredients: lidocaine 2% gel, mylanta (a solution of magnesium hydroxide and aluminium hydroxide), and dicyclomine. She then left the clinic accompanied by her family. Thirty minutes later, her family rushes back to the clinic and she is found to be gasping for air, sobbing and straddling between consciousness and unconsciousness. A pulse oximeter is quickly attached. As the patient is being taken out of the car she loses consciousness and stops breathing. Her heart rate is still present, and her O_2 saturation level drops from 98% to 85%. Cardiopulmonary resuscitation (CPR) is immediately performed in the back seat of the car and an ambulance is called. Patient has become responsive and started breathing on her own within 10 minutes. By the time the ambulance arrived, she is able to answer questions and appears comfortable. At the emergency department (ED), patient has an elevated D-Dimer.

REVIEW OF SYMPTOMS

Vague abdominal pain, back pain, unilateral leg pain resembling sciatica.

Physical Examination

- *Homan's sign*: Positive—patient was not letting examiner to touch lateral leg.
- *On dorsiflexion*: Calf pain is present.

- *Lungs*: Clear to auscultation bilaterally, no obvious findings.
- *Abdominal*: Periumbilical tenderness on palpation.

Laboratory Test

- D-Dimer positive.

Imaging

- Spiral computed tomography (CT) scan pending.

Diagnosis

The D-Dimer and acute shortness of breath (SOB), dyspnea, alongside unilateral leg pain strongly suggests pulmonary embolism. Sometimes PE is very difficult to diagnose unless you have high index of suspicion. Panic attacks, MI, acute pneumonias, and pneumothorax may have overlapping symptoms.

WORK UP

Differential Diagnosis

- *Cardiac:* Acute coronary syndrome, aortic stenosis, atrial fibrillation, cardiomyopathy, congestive heart failure, cor pulmonale, mitral stenosis, myocardial infarction, myocardial ischemia, pericarditis and cardiac tamponade, sudden cardiac death, superior vena cava syndrome, and cardiogenic shock

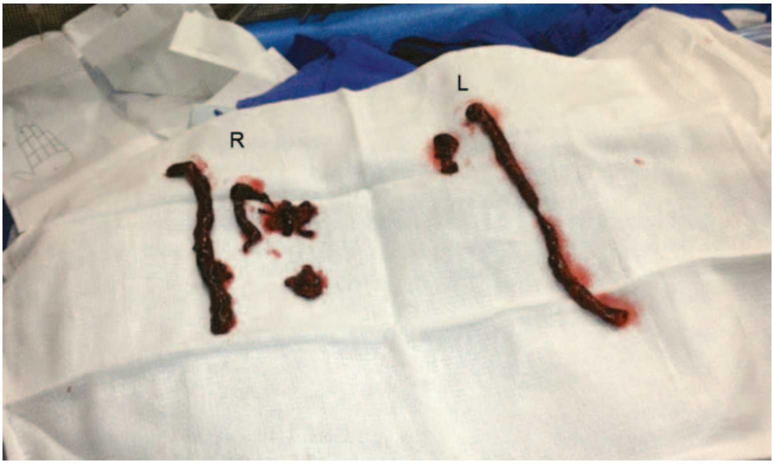


Fig. 1: Massive Pulmonary Embolism

Source: Image courtesy of Michael C. Bond, MD Assistant Professor Residency Program Director Department of Emergency Medicine University of Maryland School of Medicine

- *Pulmonary*: Acute respiratory distress syndrome, chronic obstructive pulmonary disease, pulmonary edema, emphysema, fat embolism, extrinsic allergic alveolitis, lung arteriovenous malformation, pneumothorax, pulmonary edema, noncardiogenic, pulmonary hypertension
- *Psychological*: Anxiety disorders
- *Neurologic*: Central apnea
- *Homan's sign*: Dorsiflexion of leg while the knee is at 90° angle causes calf pain. Relatively useless, low negative predictive value (NPV) and positive predictive value (PPV).
- *Chest X-ray*: Initial workup in patients complaining of dyspnea and tachypnea includes a CXR to rule out pneumonia, tuberculosis, malignancies and pneumothorax. If CXR is abnormal but not diagnostic for any of the above, a helical CT is required. A normal CXR should be followed up with a ventilation/perfusion (V/Q) scan.
  - A typical chest X-ray will show a wedge-shaped, triangular opacity with an apex pointing toward the hilum or decreased vascularity (Westermark sign).
  - Pulmonary artery will be prominent.
- *Compression ultrasound*: This is a Doppler ultrasound of the deep veins in both that looks for thrombi.
- *D-Dimer test*: This is a blood test that measures the level broken down fibrin products that are covalently bound. A positive result indicates active thrombotic events. It is currently used to rule in DVTs, PE and disseminated intravascular coagulopathy (DIC). Normally a D-Dimer has a very high-negative predictive value. However, if the PE is very likely and the patient has a strong story, a negative D-Dimer cannot rule out a PE (Table 1).
- *CT angiography*: Spiral CT (Figs 3A and B) can be very sensitive in showing thrombi in the lungs. PPV and NPV approach higher than 95%. However, they may show smaller thrombi with unclear clinical consequences (Figs 2 and 4).
- *Ventilation/Perfusion scan*: This involves a ventilation phase in which a patient inhales radioactive nucleotide gases and a perfusion phase in which the patient is injected with a radioactive albumin. A gamma camera is used during each phase to show the level of these materials in the lungs. A V/Q scan is used if CTs are unavailable or contraindicated.
- *Pulmonary angiography*: This is the gold standard, but it is invasive and expensive.

Therapies

- *Acute anticoagulation*
  - *Unfractionated heparin*: 80 U/kg then change by 18 U/kg/hr to get to partial thromboplastin time of 65–80 seconds.

Table 1 : Well's criteria	
Variable	*Point*
Clinical symptoms of DVT (leg tenderness and unilateral swelling)	3
No alternative diagnosis	3
Heart rate higher than 100 beats/minute	1.5
Immobilization or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Patient Risk	
Low risk (<10%)	< 2
Moderate risk (10–40%)	2–6
High risk (40–80%)	> 6
Likelihood of Physical Examination	
PE likely	> 4
PE unlikely	< or = 4

Source: Reproduced with permission from Wells PS, Anderson DR, Rodger M, et al. Derivation of a simple clinical model to categorize patients' probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer. Thromb Haemost. 2000;83(3):416-20. Available online from (http://www.schattauer.de/en/magazine/subject areas/journals a-z /thrombosis-and-haemostasis/contents/archive/manuscript/2372.html)

Fig. 2: Helical CT shows an intraluminal clot in anterior segmental artery in left upper lung[1]

Source: Garg K. Acute Pulmonary Embolism (Helical CT). eMedicine. In: Judith K. Amorosa et al. 2008. MedScape. 2009. Available online from http://emedicine.medscape.com/article/361131-overview

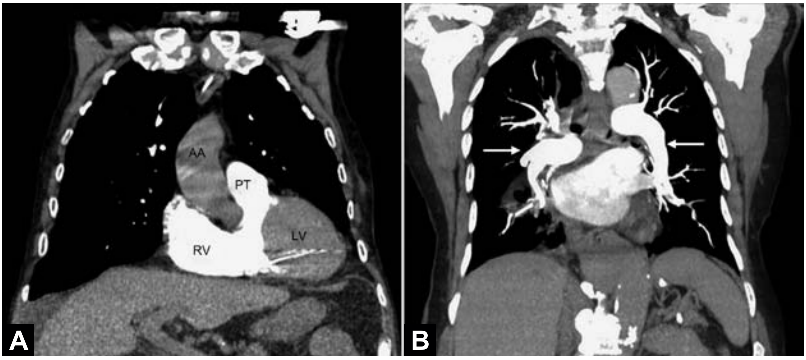
 - *Low molecular weight heparin (enoxaparin)*: It is preferred choice unless patient is obese or has renal failure.
- *Thrombolysis*
 - *Tissue plasminogen activator*: 100 mg over 2 hours, reserved for massive PE.
- *Catheter-directed therapy*
 - Fibrinolytic and thrombus fragmentation/aspiration.
 - Used in extensive PE and in those patients in whom systemic thrombolysis is contraindicated (high risk for bleeding).
- *Thrombectomy*
 - Reserved for patients with massive or extensive PE at large centers that cannot tolerate systemic thrombolysis.
- *Long-term anticoagulation*
 - *Warfarin*: Start at the same time as heparin.
 - Can be used as single agent for proximal DVT and uncomplicated PE.
 - Used alongside enoxaparin for PE associated with cancer.
- *Inferior Vena Cava filter*
 - Used for DVT at risk of embolization.

MASSIVE PULMONARY EMBOLISM

Definition: Sustained hypotension, SBP < 90 mm Hg (at least 15 min), inotropic support, pulseless, persistent profound bradycardia (see Fig. 1).

Thrombolytics

- *Thrombolytics-Benefits*: Rapid resolution of sypmtoms, stabilization of resp/cv function, cocharane review: reduce mortality and recurrence.
- Should not be delayed for patients with hemodynamic compromise.
- 3 agents FDA approved streptokinase, urokinase, alteplase.
- *Alteplase*: Boluse dose 10 mg, Infusion 90 mg over 2 hrs. Peripheral IV.
- Heparin infusion should be paused.



Figs 3A and B: Pulmonary embolism CT

Source: Available online from http://www.ceessentials.net/images/pulmonaryEmbolus/image018.jpg

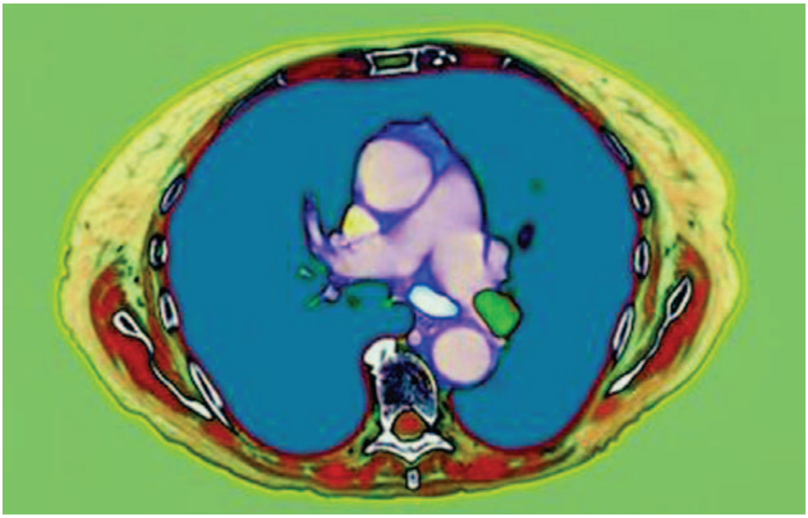


Fig. 4: Pulmonary embolism colored CT

Source: Available online from http://www.images.cpcache.com/merchandise/514_400x400_NoPeel.jpg?region=name:FrontCenter,id:71660204,w:16

REFERENCE

1. Garg K. Acute Pulmonary Embolism (Helical CT). *eMedicine*. In: Judith K. Amorosa et al. 2008. Medscape. 2009. Available online from http://emedicine.medscape.com/article/361131-overview

ADDITIONAL READING

1. Michael C Bond, MD Assistant Professor Residency Program Director Department of Emergency Medicine University of Maryland School of Medicine

Case Study 10: Pulmonary Contusion

CASE HISTORY

An 8-year-old boy is presented to the emergency department (ED) after being hit by a car. The driver was attempting to stop when the boy rode in front of the car on his roller skates. The boy was struck with the vehicle at low speed while the vehicle was attempting to stop. The boy struck the car and then the ground. He was wearing his helmet which struck the ground with him. He did not lose consciousness at any point. Primary survey reveals intact airway, breathing and circulation (ABCs) and mental status. Secondary survey reveals multiple contusions and abrasions on his arms and legs. The boy is holding his right (R) arm close to his chest. Vitals are initially unremarkable except for tachycardia to 140 beats/minute. Fluids and analgesia are started. X-ray of the R-forearm reveals an R-forearm fracture. Distal pulses and sensation are intact. While waiting for further orthopedic follow-up the boy begins to complain of difficulty in breathing. His oxygen saturation continues to slowly drop down from 100% to 97%. He is started on O^2 by nasal cannula, but continues to worsen. Immediate chest X-ray (CXR) is ordered and reveals infiltrates on the left (L) lower lung fields. No rib fractures or pneumothorax is present. What is the diagnosis? What management steps should be considered?

DISCUSSION

Pulmonary contusion typically occurs in younger patients without completely ossified ribs. It may occur with or without the presence of flail chest or rib fractures.[1,2] Pulmonary contusion is a common potentially fatal injury (Fig. 1). Clinically, respiratory failure may present insidiously. Contusions present with pulmonary infiltrates and hemorrhage into pulmonary tissue.

Consider intubation for patients with significant hypoxia. Pre-existing conditions, such as chronic obstructive pulmonary disease (COPD) or renal failure, may warrant earlier intubation. If patients with these conditions are transferred to your institution, they will require mechanical intubation. Every patient needs to be monitored for oxygen saturation, serial electrocardiograms (ECGs), arterial blood gases (ABGs) and suctioning, if needed. Basic laboratory tests values such as complete blood count (CBC), troponin, chest CT or chest X-ray should be repeated as needed.

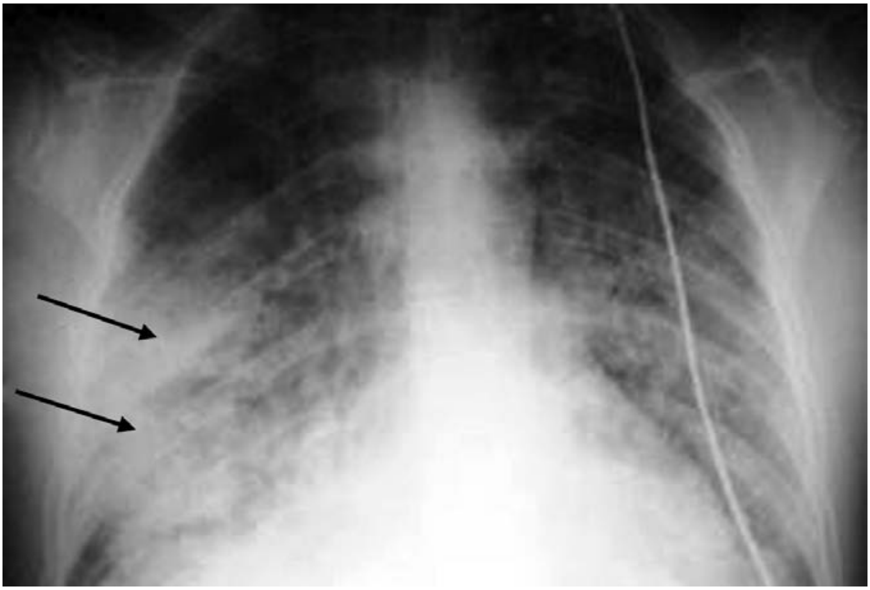


Fig. 1: White-out appearance with rib fractures (arrows)
Source: http://en.wikipedia.org

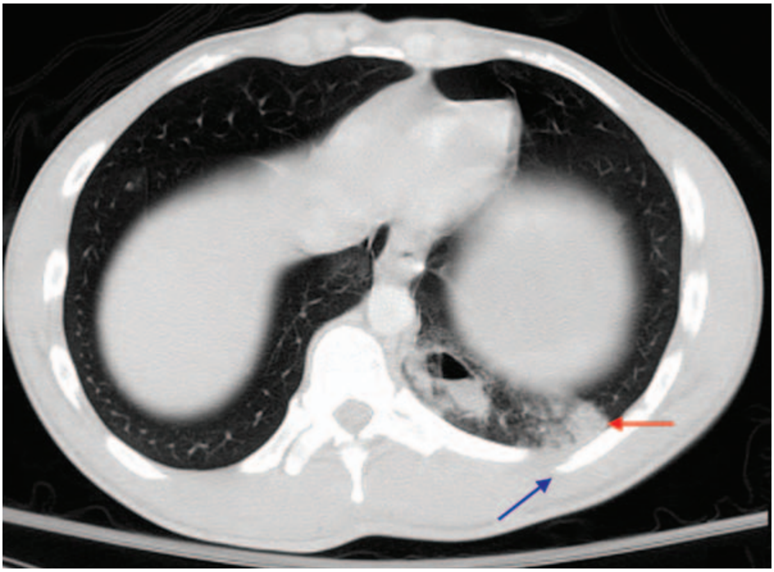


Fig. 2: CT chest with rib fractures (yellow arrow) and pulmonary contusion (red arrows)
Source: NIH & Medscape

Flail chest can often be seen with pulmonary contusions. Flail chest presents with multiple rib fractures and paradoxical breathing, which is when the chest bulges out on expiration and caves in on inspiration. Flail

chest presentation is very sensitive to fluid overload. So in treatment, we must restrict fluid intake.

Chest contusion may present with a white-out appearance on chest X-ray. We will need to continue monitoring because 50% of patients after trauma show on signs or symptoms of pulmonary contusion.

Treatment

- Judicious use of fluids. Use colloids for resuscitation if needed, avoid crystalloids.
- Diuretics
- Intubation with PEEP and cardiopulmonary monitoring in ICU.
- Early intervention of a pulmonologist will have a better outcome.

REFERENCES

1. Mary C Mancini. Blunt Chest Trauma. Available online from http://emedicine.medscape.com/article/428723-overview#a7
2. Thoracic trauma. ATLS. p. 94.

Case Study 11: Tension Pneumothorax

"Preparation is everything"

—Badar M Zaheer

CASE HISTORY

A 17-year-old boy was crushed between the garage door and the bumper of a car while his younger brother started the car and was trying to backup. This was his first driving experience without supervision. He was presented to the emergency department in a semirigid cervical collar and immobilized on a long back board on 10 L/min of oxygen via mask.

Physical examination shows a child who is alert but in severe distress. His vital signs are temperature 98.8°F, pulse rate 144 beats/minute, respiratory rate 40 breaths/minute and blood pressure 150/70 mm Hg. His airway is patent but respirations are rapid and shallow and he is cyanotic. There is decreased movement of the left hemithorax and there are absent breath sounds on left. There is mild subcutaneous emphysema over the left chest wall and neck but no tracheal deviation. Cardiac examination reveals tachycardia with a regular rhythm and jugular venous distension.

Chest X-ray is consistent with a left-sided pneumothorax.

PNEUMOTHORAX

A pneumothorax is a collection of air around the lungs which subsequently puts pressure on the lungs so that it cannot expand during inspiration. Pneumothorax presents initially as respiratory distress, often with tachypnea and shallow breathing. On physical examination, there will be decreased movement of the hemithorax and absent breath sounds on the affected side. In more severe cases, there may also be subcutaneous emphysema of the chest wall and neck on the injured side as well as tracheal deviation toward the unaffected side.

TREATMENT

The first step in management in a person with a pneumothorax is evaluation of the airway, breathing and circulation (ABCs). Determination of airway patency and evaluation of the quality of the breathing should be done. Administration of oxygen via nasal cannula or mask is the first step. If the patient continues to experience respiratory distress despite supplemental oxygen, intubation should be considered. It is critical to recognize a pneumothorax quickly in order to initiate treatment and relieve the pressure. Once the patient is stabilized, immediate decompression is needed; the first step is to insert a large caliber needle into the 2nd intercostal space on the

midclavicular line of the affected pneumothorax (Fig. 1). An immediate release of air will occur. A chest tube is inserted with a 20–26 French chest tube in the 4th or 5th intercostal space in the anterior axillary line (Fig. 2). A chest X-ray should be repeated at this time to confirm chest tube placement and improvement of the pneumothorax. The patient should also be placed on continuous pulse oximetry to monitor oxygen levels. Circulatory status should be maintained with two large bore IVs and normal saline infused to maintain intravascular volume.

Remember:

- Chest tube should be directed above for air (pneumothorax) or below for blood (hemopneumothorax) and connected to drainage canisters shown below (Fig. 3).
- "A" for Air and "B" for Blood.
- Always remember not to insert the needle or chest tube below the rib to avoid bleeding from intercostal vasculature.

Fig. 1: Angiocath is inserted at a 90 degree angle and advanced till air escaping is heard
Source: http://www.civiliandefenseforce.com/needledecompression.html

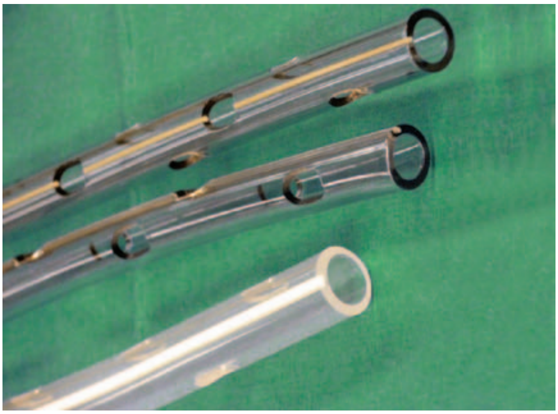


Fig. 2: Chest tube
Source: http://en.wikipedia.org/wiki/chest_tube

Fig. 3: Drainage canisters pleur evac
Courtesy: http://en.wikipedia.org/wiki/Chest_tube

ADDITIONAL READING

1. http://www.ncbi.nlm.nih.gov/pubmed/16825880
2. http://en.wikipedia.org/wiki/Hemopneumothorax

Case Study 12: Hemopneumothorax

CASE HISTORY

A 15-year-old boy was crushed between a wall and the back of a van that was reversing. He is lying on the parking in severe respiratory distress. He is breathing at 40 breaths/minute, labored and shallow. He is alert and moves all of his extremities. The patient's blood pressure is 160/70 mm Hg and he is transported on a rigid board with a C-collar on with supplemental oxygen.

PREHOSPITAL TREATMENT

On arrival to the emergency room, the patient is in severe respiratory distress. He has absence of breath sounds on the left side of chest and tracheal shift to the right side.

Respiration

Crepitus is heard over left chest wall and is extending up to the neck.

Abdominal Findings

Abdomen is distended and tender on palpation. Bowel sounds are absent.

Imaging

Chest X-ray findings includes left tension pneumothorax, right lung contusion and acute gastric dilatation (Fig. 1).

TREATMENT

- *Primary survey*: Carrying out the airway, breathing, circulation, disability and exposure (ABCDE) is important. Remove all clothing.
- A = Assess the airway (respiratory distress at 40 beats/minute).
  - Airway suction and lifting of chin is done
  - If pulse oximeter has saturation of 80%, switch to nonbreathing mask (cyanosis present)
  - If patient becomes increasingly lethargic, anxious, and shows increased respiration rate, then assess the breathing adequacy.
- Intubation is not required at this point, but chest tube placement is our priority.

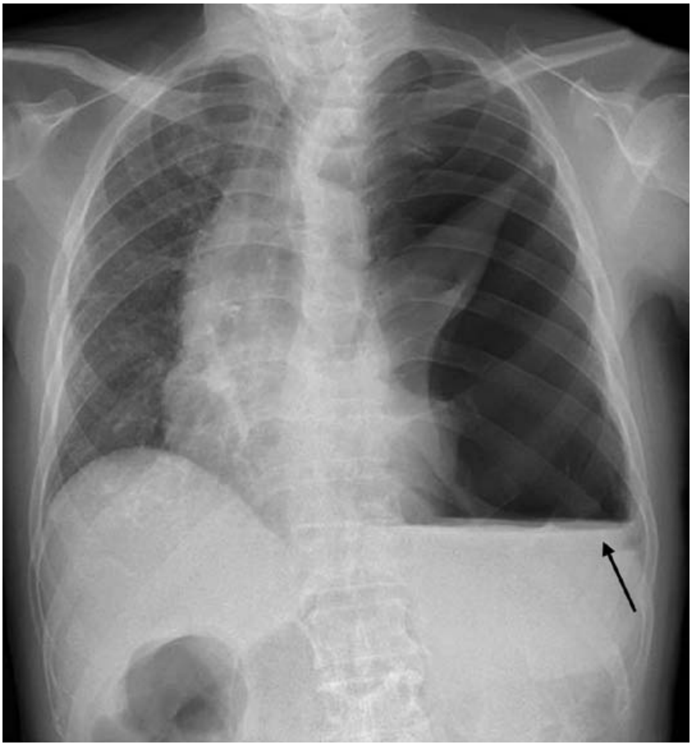


Fig. 1: Chest X-ray for hemopneumothorax blood/fluid level (arrow)
Source: Postgrad Med J© 2012 The Fellowship of Postgraduate Medicine

Physical Findings

It includes the following:
- Tension pneumothorax on left chest wall.
- Hyper-resonance and absence of breath sounds on left chest.
- Subcutaneous emphysema on left side.
- Tracheal deviation on the right chest wall. (opposite of the pneumothorax)

Difficulty in Using Bagwell Mask

Treatment: A large caliber needle is inserted over the second intercostal space at midclavicular line (always put needle on top of rib). You hear a gush of air and the patient immediately feels better.

Improvement of Symptoms

- Child becomes more alert, color improves, breath sounds are less diminished on the right chest wall and trachea begins to return to midline.

- This procedure is followed by a chest-tube insertion to replace the needle.
- A chest-tube (size 24 french) is inserted and chest X-ray (CXR) is ordered.
- Saturation improves and arterial blood gas (ABG) comes back to normal.
- *Circulation*: Warm-crystalloid solution is started in both arms.
- Cardiac monitor shows heart rate (HR) of 120 beats/minute.
- Because of possibility of abdominal trauma, a rectal examination was performed. A urinary catheter was placed after the rectal examination.
- Maintenance fluid is infused at a rate of 1 mL/kg/hr. Patient is 40 kg so 40 ml/hr is used.
- Glasgow coma scale (GCS) = 12 (Child is opening his eyes, pupils are equal and reactive, and is obeying commands).

ADDITIONAL READING

1. http://www.ncbi.nlm.nih.gov/pubmed/16825880
2. http://en.wikipedia.org/wiki/Hemopneumothorax

Case Study 13: Spontaneous Pneumothorax

"Listen if you can, bear listening...keep silence where visions are expressed"

—Rumi

CASE HISTORY

A 29-year-old male is presented to the emergency department (ED) with difficulty in breathing and a dull-chest pain. The patient reports that the symptoms started all of a sudden 7 hours prior to coming in. At the time the chest pain on the right chest was more like a "stabbing" and now it has a vague and dull sensation. The pain is worse with inspiration. The patient has no past medical history except for smoking one packet per day of cigarettes for the last 10 years. On physical examination, his vital signs are blood pressure 150/80 mm Hg, heart rate 110 beats/minute, respiratory rate 40 breaths/minute and O_2 saturation of 90%. Patient is a tall thin appearing male in significant respiratory distress. He is started on nonrebreather oxygen mask at 12 L and intubation cart is prepared. Electrocardiography (ECG) and chest X-ray (CXR) are ordered.

DISCUSSION

This patient is in severe respiratory distress and action must be taken quickly in order to reverse the effects. The differentials include pleurisy, pulmonary embolism, myocardial infarction, pericarditis, asthma, pneumonia and pneumothorax. The first goal of treatments should be to stabilize the airway, breathing and circulation. In severe cases, early definitive management should include needle aspiration or chest-tube placement.

Spontaneous pneumothorax is usually confirmed by upright chest X-ray (Figs 1 and 2).[1] On CXR, a white-visceral pleural line with the absence of vessel markings peripheral to this line must be searched. If the lateral width is greater than 10%, a diagnosis of pneumothorax is made. The most sensitive positive for detecting a pneumothorax is the left lateral decubitus with the least sensitive being supine. Even higher sensitivity may be obtained by obtaining both inspiratory and expiratory films. Computed tomography (CT) may be used to further classify or detect hard to see pneumothoraces. Furthermore, CT may detect underlying causes for secondary pneumothoraces. Bedside ultrasounds are equally effective in diagnosis.

This management differs from a suspected tension pneumothorax which is always a medical emergency and in fact should be detected by CXR or it may be too late to intervene. Tension pneumothorax must

be suspected when the patient is hemodynamically unstable or when contralateral tracheal or mediastinal deviation is present (Table 1).
One goal of treatment after stabilization is prevention since the overall recurrence rate is estimated at 5%. A current mainstay of treatment is video-assisted thoracoscopy (VATS) with an aim to excise the associated bullae or perform guided pleurodesis. Talc has frequently been indicated as a sclerosing agent. However, patients with small pneumothoraces, less than 20%, may be observed with repetition of chest X-ray. Regardless, definitive management is usually done after the first recurrence except in cases with high-risk occupations such as divers or pilots.

Table 1: The pathophysiology, presentation and progression diagnosis and treatment of spontaneous and tension pneumothorax

	Spontaneous pneumothorax	*Tension pneumothorax*
Pathophysiology	• Primary = Rupture of small blebs near apex of lungs • Secondary = Due to chronic obstructive pulmonary disease (COPD) (most common), pneumonia, bronchogenic carcinoma, mesothelioma, sarcoidosis, tuberculosis, cystic fibrosis	• Usually traumatic event causes the formation of a check valve mechanism that lets air into the pleural space but it does not let it out
Presentation and progression	• Pleuritic chest pain that starts as sharp and turns dull (90%) • 5% of patients have no symptoms and can delay treatment up to a week	• Rapid progression of dyspnea and tachypnea • Emergency
Diagnosis	• Chest X-ray (Fig. 1)  **Fig.1:** Tracheal deviation towards pneumothorax	• Tracheal deviation away from pneumothorax • CXR (Fig. 2)  **Fig. 2:** Tracheal deviation away from pneumothorax
	CT can be used if CXR is not conclusive	

Treatment	• 100% O_2: Increases the absorption of pneumothorax and should be administrated to all patients with pneumothorax. • Observation alone in asymptomatic patient with a small (< 3 cm between lung and chest wall on CXR) pneumothorax. However, a repeat CXR is necessary to demonstrate the stability of the condition, which requires close monitoring.	• 100% O_2 • Needle aspiration needed in patient with rapidly deteriorating condition and more than 3 cm between lung and chest wall. • Needle aspiration can be done using a large-bore angiocatheter needle. The needle is introduced in the second intercostal space of midclavicular line. The catheter is left in place and attached to a three-way stopcock and a large syringe. Air is aspirated until resistance is met or the patient experiences significant coughing. • Repeat CXR is done immediately after aspiration and again in 4–24 hours to document re-expansion of the lung. • If the pneumothorax fails to resolve with aspiration, a chest tube should be placed.

REFERENCE

1. Ferri FF: (2012) Ferri's Clinical Advisor 2013, 1st edit ion. Elsevier: Mosby. [online] Available online from www.mdconsult.com/books/page.do.

Case Study 14: Deep Vein Thrombosis

CASE HISTORY

Patient is a 43-year-old female who is presented with right leg pain associated with redness and swelling of the calf area. Patient has a history of a similar episode 2 years ago for which she took coumadin until 4 months ago. She took oral contraceptives for a few years in her 30s. She has no other medical problems except the history of *deep veinnous thrombosis* (DVT) in the past. She is not currently taking any medication. She has no recent immobilization. No symptoms of fever or shortness of breath were noticed. On physical examination, she has a temperature of 98.6°F, pulse rate 80 beats/minute, respiratory rate 18 breaths/minute, and blood pressure 120/70 mm Hg. There is tenderness in the right lower extremity and the calf is slightly warm to palpation as compared to the left leg. Homan's sign, pain in the calf muscles with forced dorsiflexion of the foot, is absent. Lab workup shows complete blood count (CBC) within normal limits and D-dimer is elevated. Doppler ultrasound shows a high clinical probability of DVT.[1]

DISCUSSION

In cases of leg pain and swelling several diagnoses should be considered, such as congestive heart failure (CHF) exacerbation, cellulitis, venous stasis without thrombosis, musculoskeletal injuries, vasculitis as well as DVT. Given the patient's history of DVT in the past, unilateral leg pain and swelling, elevated D-dimer, and absence of signs of heart failure or injuries she likely has a recurrent DVT.

The classical signs and symptoms of a DVT include calf or leg pain, redness, swelling and warmth (Fig. 1). Unfortunately these are present in only about 50% of patients who present with a DVT. Homan's sign is an unreliable diagnostic test for DVT. For these reasons, criteria have been developed to determine the probability of whether or not a patient has a DVT, of which the most universally used is the Well's criteria.[2] The following Box 1 shows the list of Well's criteria and the scoring system.

Box 1: Well's score or criteria (Possible score – 2 to 9)

1. Active cancer (treatment within last 6 months or palliative): + 1 point
2. Calf swelling ≥ 3 cm compared to other calf (measured 10 cm below tibial tuberosity): + 1 point
3. Collateral superficial veins (nonvaricose): + 1 point

Fig. 1: Venous eczema

Source: Available online from http://www.healthhype.com/deep-venous-thrombosis leg-vein-clot-dvt-pictures-symptoms.html [Accessed June, 2012].

Figure courtesy: da Silva SF, Dermatology Atlas

4. Pitting edema (confined to symptomatic leg): + 1 point
5. Previous documented deep vein thrombosis: + 1 point
6. Swelling of entire leg: + 1 point
7. Localized pain along distribution of deep venous system: + 1 point
8. Paralysis, paresis, or recent cast immobilization of lower extremities: + 1 point
9. Recently bedridden ≥ 3 days, or major surgery requiring regional or general anesthetic in past 4 weeks: + 1 point
10. Alternative diagnosis at least as likely: – 2 points

A score of 3 or more indicates a high probability of DVT, a score of 1–2 indicates at moderate probability, and a score of 0 or less indicate a low probability. The need for further workup is determined by the score on these criteria. D-dimer assay is one option for testing; however, this test has a very low sensitivity of about 80% and therefore is not useful in ruling out DVT. Duplex ultrasonography has a sensitivity of 97% and specificity of 94% and is considered a reliable method of diagnosis. For patients with a high probability of DVT but a negative Doppler, the test should be repeated in 1 week. After two consecutive negative Doppler ultrasounds 1 week apart, the risk of symptomatic DVT or pulmonary embolism (PE) within the next 3 months is less than 1%.

Treatment includes anticoagulation to prevent extension of the clot and decrease risk for PE and allow the natural fibrinolytic system to function. The treatment should begin with a low-molecular weight heparin (LMWH), such as dalteparin, enoxaparin or tinzaparin, with a dose given every 24 hours. Unfractionated heparin can be used when LMWH is contraindicated and lepirudin can be used for patients with a history of heparin-induced thrombocytopenia. Warfarin should be started concurrently at a dose of 5 mg/day with a goal International Normalized Ratio (INR) of 2.0–3.0. Bridging with heparin should be continued until the aimed INR is reached. In cases in which anticoagulation is contraindicated, inferior vena cava (IVC) filter can be placed to prevent the development of a PE. Most patients can be discharged safely with close follow-up if a dose of LMWH is given except for patients with complicated cases or patients with lack of follow-up.

REFERENCES

1. Kabrhel C. Peripheral vascular disorders. Emergency Medicine Manual, 6th edition. pp. 166-8.
2. Well's criteria used. No copyright needed.

Case Study 15: Asthma

CASE HISTORY

A 32-year-old man who has history of asthma is presented to emergency department (ED) in severe respiratory distress. He was wheezing and using accessory muscles of respiration. His vital signs are blood pressure 120/90 mm Hg, heart rate 120 beats/minute, respiratory rate 40 breaths/minute, temperature 97°F, O_2 saturation 88% on room air (RA). This patient was brought to emergency room (ER) by ambulance. His O_2 saturations remain 88% on RA. Even with beta-2 agonist, the patient still remains in respiratory distress and with ipratropium treatment the saturation improves slightly, patient stating 90% O_2 saturation. Initial peak flow was less than 200 L/min. Dexamethasone 4 mg IV is given at this time.

DISCUSSION

This patient is obviously in respiratory distress and needs immediate coordination from physicians, respiratory therapist and nursing staff. The objective of the treatment is to prevent the patient from going to respiratory failure. Asthma is a complex disorder that results from airway inflammation, airflow obstruction and bronchial hyper-responsiveness. Many factors may contribute to the hyper-reactivity including environmental allergens, exercise, occupational exposure, emotional factors, gastroesophageal reflux disease (GERD), chronic sinusitis and many more. The differential is broad and includes vocal cord dysfunction, tracheal and bronchial lesions, foreign bodies, pulmonary migraine, congestive heart failure, diffuse panbronchiolitis, aortic arch anomalies, sinus disease, cystic fibrosis and pulmonary embolism.

When assessing asthma, it is important to determine precipitating factors: rapidity of onset, associated illness, number of exacerbations in the last year, need for ED visits, hospitalizations, ICU admissions, intubations, and missed days from work or school/activity limitation. Asthma presents with wheezing as a very common symptom.[1] Wheezing may vary from mild, only at the end of expiration to severe, lasting throughout expiration. In extremely severe cases, wheezing may be absent from severe outflow obstruction. Other symptoms include cough or chest tightness. With worsening episodes, patients will be unable to lie flat and may even be hunched forward, talk is of short words or phrases, and may be agitated. Use of accessory muscles may be seen. Respiratory rate is high and often greater than 30 breaths/minute. Heart rate is generally more than 120 beats/minute.

Initial evaluation should include electrocardiography (ECG), pulse oximetry and chest X-ray. In cases of asthma, chest X-ray may indicate hyperinflation, but will also be used to investigate other sources for respiratory distress. Peak expiratory flow may be used easily in the ED to compare to patient's baseline and response to treatment. Optimization of care should be done as an outpatient basis.

In the ED, the most common treatment will be the use of beta 2 agonists which may be administered via inhaled or nebulized forms. The main side effect is tachycardia, which is well tolerated in children, but must be carefully monitored in adults with comorbid illnesses.

Ipratropium may also be used as a bronchodilator. Steroids should be used in moderate to severe cases. There is no explicit benefit of using oral versus intravenous steroids other than the possible ease of administration. The mainstay of treatment is to use enough bronchodilators to allow the steroids to take effect to diminish the bronchial inflammation. Heliox is also shown to provide relief to patients in the ED. Intubation should be considered in patients who fail to respond to treatment.

Considerations for admission should be made on a case-by-case basis given history of frequent exacerbations, comorbid illness and response to treatment. In severe cases, ICU admission should be considered for patients who may be somnolent, have significant hypoxemia, hypercapnia or who require intubation. Patients who are on RA, room air, maintaining their saturation, and are no longer symptomatic while walking may be discharged home.

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Case Study 16: Acute Respiratory Distress Syndrome

CASE HISTORY

A 78-year-old Caucasian male was noted to be very lethargic for the past 4 days by his family members. His family called Emergency Number 911 and brought him to nearest hospital by emergency medical services (EMS). Patient was on nonrebreather (NRB) mask during transport to the nearest hospital. His vital signs are heart rate 123 beats/minute, temperature 101.2°F, respiratory rate, 40 breaths/minute and shallow, blood pressure 70/50 mm Hg, O_2 saturation 78% on NRB mask. His past medical history (PMH) includes asthma, hypertension and diabetes mellitus. Upon arrival, patient is intubated by emergency department (ED) physician and placed on 100% fraction of inspired oxygen (FiO_2), he is given 2 L fluid boluses. Laboratory data includes WBC count of 22,000/µL, bands 5%, lactic acid 9 mmol/L and sputum cultures were sent. Portable chest X-ray (CXR) shows left lower lobe (LLL) infiltrates and CT scan of head negative for acute intracranial process or bleed. CT scan of chest shows negative results for pulmonary embolism. Patient is admitted to intensive care unit (ICU) for acute respiratory distress syndrome (ARDS) from pneumonia. He is placed on sepsis protocol as per ICU routine and he continued to receive 1 L fluid bolus and 0.09 normal saline (NS) placed at 150 cc/hr after. He is given injection Rocephin 2 gms intravenous piggyback [IV short-term infusion (IVPB)] q 24 hrs. Tylenol supplement is used for fever. Ventilator settings in ICU are given as follows: Assist control (A/C) mode = 12 hours, Tidal volume (TV) = 450 cc, Pressure support = 10 cm H_2O, Positive end-expiratory pressure = +5 cm H_2O, Fraction of inspired oxygen (FiO_2) = 100% and taped 23 at the lip. Patient is given propofol drop (gtt) for Richmond agitation-sedation scale (RASS) of –2 or 15 mcg/kg/min.

DISCUSSION

Acute respiratory distress syndrome includes an abrupt onset of diffuse lung injury accompanied by severe hypoxemia and bilateral pulmonary infiltrates.[1] Having a PaO_2/FiO_2 of less than 200 is suggestive of ARDS. Every year there are 45–75 cases/100,000 people. Risk factors includes severe infection, aspiration, shock, lung contusion, nonthoracic trauma, toxic inhalation, near drowning and multiple blood transfusions.

Pathophysiology includes an inflammatory response consisting of an acute exudative phase, followed by a fibrosing alveolitis phase lasting 1–2 weeks during recovery, and finally resolution requires anywhere between 6 and 12 months.

Historical factors include absence of heart disease and history of any precipitating event. Physical examination demonstrates tachypnea, tachycardia, respiratory distress, lethargy, obtundation, flat neck veins, hyperdynamic pulses, physiologic gallop, absence of edema, moist and cyanotic skin, and manifestations of underlying disease. Arterial blood gas (ABG) will show severe hypoxemia. As described above, PaO_2/FiO_2 will be less than 200. Electrocardiography (ECG) may show sinus tachycardia or nonspecific ST changes. Pulmonary artery wedge pressure will be less than 15 mm Hg, and cardiac index will be greater than 3.5 L/min/m^2. Initial imaging should include CXR and CT chest followed by serial CXRs. CXR will show fluffy bilateral infiltrates and CT chest will show diffuse interstitial opacities and bullae. Differential diagnosis includes left ventricular failure, interstitial and airway disease, veno-occlusive disease and mitral stenosis.

Patients with ARDS should be admitted to the ICU. Overall treatment goal is supportive with management directed at the underlying cause. Patients should be given supplemental oxygen and placed on ventilatory support. Ventilation settings should include lower tidal volumes and optimization of PEEP. Other treatment goals are directed toward sepsis protocol including use of ionotropic agents such as dobutamine to maintain blood pressure support. Corticosteroid use is controversial, but sustained therapy in severe ARDS may be beneficial. Inhaled nitric oxide is no longer indicated. Some evidence suggests that pulmonary surfactant may be used in neonatal cases. Bronchodilators may be helpful while recovering. Data for antioxidants is controversial. Other supportive measures include deep vein thrombosis prophylaxis, ulcer prophylaxis and parenteral nutrition. Invasive monitoring of vital signs, cardiac function, and pulmonary wedge pressure is also controversial. Large clinical trials have called the utility of these measures into question.

Overall prognosis is poor with a mortality rate of 47%. Those that do survive often have multiple complications such as pulmonary lung disease, oxygen toxicity, barotrauma, superinfection and multiorgan dysfunction (Figs 1 and 2).

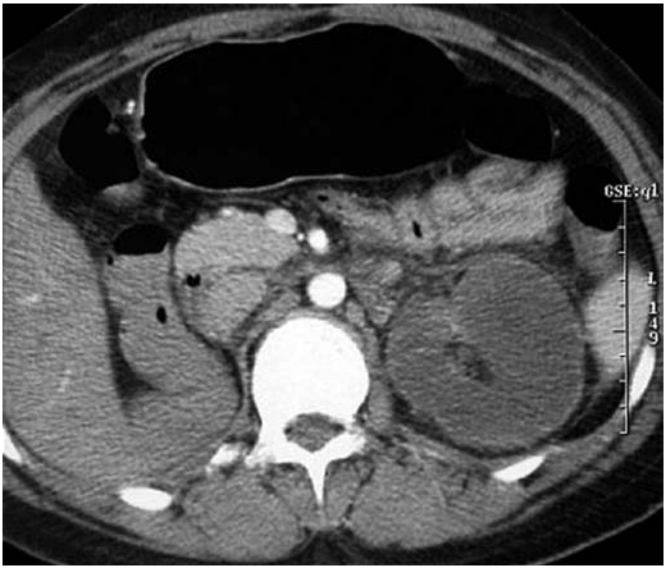

Fig. 1: CT andomen in ARDS

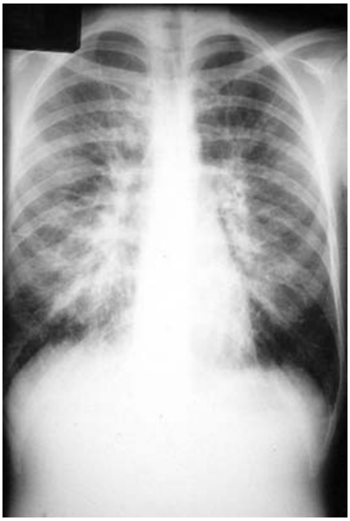

Fig. 2: Marked interstitial edema with hilar indistinctness, Kerley B lines, in hantavirus pulmonary syndrome (HPS)

Source: Available online from http://www.cdc.gov/hantavirus/technical/hps/clinical manifes tation.html. [Accessed June, 2012]

Figure courtesy: Ketai DL

REFERENCE

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CARDIOVASCULAR

Case Study 17: Acute Coronary Syndrome—ST-Segment Elevation Myocardial Infarction

CASE HISTORY

A 75-year-old man is seen in the emergency room (ER) for chest pain and lightheadedness for the past 4 hours. His past history includes hypertension (HTN), hyperlipidemia and diverticulosis. Current medications included atenolol 50 mg, aspirin 325 mg and lovastatin 20 mg. His vital signs are pulse rate of 70 beats/minute regular, blood pressure 90/70 mm Hg, and chest and cardiac examinations are normal. Electrocardiogram (ECG) showed normal sinus rhythm with diffuse ST-T wave abnormalities. Initial troponin level is within normal limits, but after 6 hours his troponin level has reached 9 ng/mL. His hematocrit is 26%. Rectal examination is hemoccult positive. Repeat ECG was ordered to determine further management (Fig. 1).

INTRODUCTION

Given that the patient has chest pain with a positive troponin, he likely has an acute coronary syndrome (ACS). ACS includes both myocardial

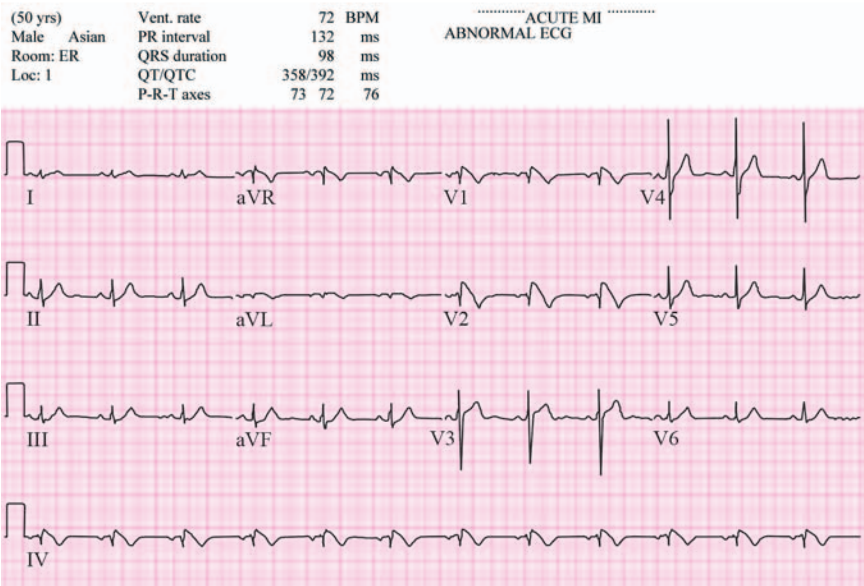
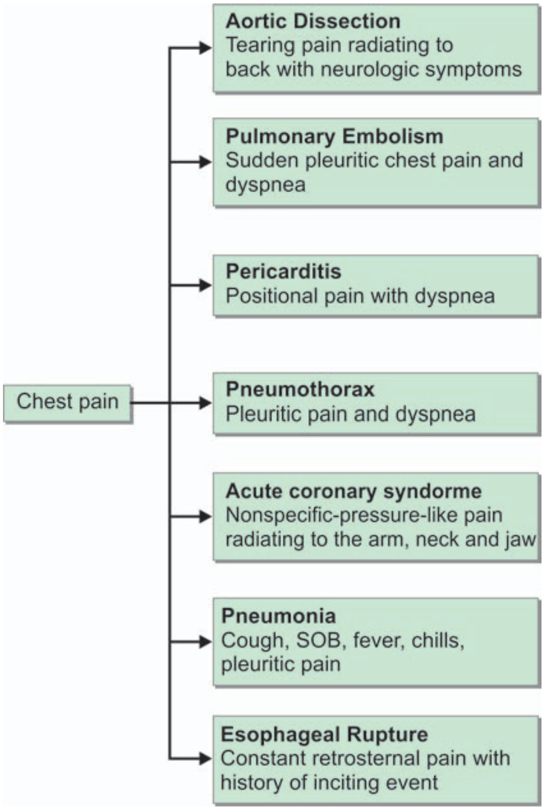


Fig. 1: ECG showing diffuse ST changes
Source: Badar M Zaheer MD

Flow chart 1: Causes of chest pain



Source: Badar M Zaheer MD

infarction (MI) and unstable angina. MI results from an imbalance between myocardial oxygen supply and demand. Oxygen supply can be disrupted by atherosclerotic lesions, vasospasm, platelet aggregation and thrombus formation. There are seven major risk factors for coronary artery disease including age, male sex, family history, cigarette smoking, HTN, hypercholesterolemia and diabetes.

APPROACH TO CHEST PAIN (FLOW CHART 1)

Chest pain may have many causes. It is important to have an approach to chest pain to first ensure that the deadly causes of chest pain are ruled out before pursuing more benign causes. The five deadly causes of chest pain are myocardial infarction, pulmonary embolism, esophageal rupture, pneumothorax and aortic dissection. Non life-threatening causes of chest pain may include panic attack, costochondritis, pneumonia, and referred abdominal pain such as gastroesophageal reflux disease (GERD) or cholecystitis.

Typical presentation for cardiac causes of chest pain includes pain that is retrosternal and is either squeezing, tightening, crushing or pressure like in nature. Typically sites of radiation include the left shoulder, jaw, arm or hand. Presentations in the elderly, diabetics, or women may not include chest pain at all but will present with extreme fatigue. In the elderly, the chief complaint may be dyspnea, diaphoresis, nausea, light-headedness, or profound weakness. This is why there should be a low threshold for ordering an ECG. Symptoms lasting less than 2 minutes or over several days are less likely to be ischemic in origin.

Physical examination may vary from normal to extremely abnormal hemodynamics. Stable angina presents in episodes anywhere from 5 minutes to 10 minutes long. Pain is worse with exertion and relieved with rest and nitroglycerin. Unstable angina has symptoms at rest. Prinzmetal angina occurs at rest and it is often associated with tobacco or cocaine use.

Aortic dissection presents with sudden onset and tearing pain that radiates to the back. Patients are typically hemodynamically unstable with either HTN or hypotension. If suspected, chest X-ray, computed tomography (CT), transesophageal echo, or angiography may be used to diagnose a dissection. Patients with pericarditis will complain of chest pain that radiates to the jaw, back or neck and is alleviated by leaning forward. ST segment changes will be diffused. Patients with pulmonary embolism with complain of sudden onset, pleuritic chest pain associated with dyspnea, tachypnea, tachycardia or hypoxemia. Chest wall tenderness supports a diagnosis of costochondritis but does not rule out acute MI. Gastrointestinal causes of pain may be associated with meals and relieved by antacids. GERD should be a diagnosis of exclusion. Cholecystitis should be considered in cases of atypical chest pain, especially in women.

All patients should be placed on oxygen, cardiac monitors, and start IV lines. Patients should be questioned about cardiac risk factors and careful history should be elicited. Serial ECGs should be obtained when considering cardiac causes of chest pain. Moderate to high-risk patients should be admitted for further observation, serial ECGs, serial enzymes and possible stress testing.

MANAGEMENT OF ACUTE CORONARY SYNDROME

Treatment depends on symptoms, history, physical and ECG findings. The overall goal is to restore perfusion and limit infarction. This may be done in several ways: mechanical fibrinolytics, angioplasty or coronary

artery bypass graft (CABG). Initially all patients should be placed on a cardiac monitor, given an IV line, and placed on oxygen. An ECG should be obtained within 5 minutes. Acetylsalicylic acid (ASA) or aspirin 160–325 mg should be given as soon as possible. Chewable form should be administered for more rapid onset. ASA has been shown to reduce death by up to 23%. Next, nitroglycerin also reduces mortality by up to 35%. Nitroglycerin may be given sublingually and repeated up to three times at 2–5 minutes intervals. If no improvement is noticed, a nitro drip should be started. The exception is the case of preload dependent right ventricular infarction. Headache is a common side effect of nitroglycerin. Morphine may be used effectively to control pain.

Beta-blockers have also been shown to reduce mortality and therefore cardioselective agents, preferably, should be used for ST-segment elevation myocardial infarction (STEMIs), recurrent ischemia, tachydysrhythmia and NSTEMIs. Relative contraindications are heart rate (HR) slower than 60 beats/minute, systolic BP less than 100 mm Hg, moderate to severe congestive heart failure (CHF), signs of peripheral hypoperfusion, pulse rate (PR) interval longer than 0.24 seconds, second- or third-degree atrioventricular block (AVB), severe chronic obstructive pulmonary disease (COPD), history of asthma, severe diabetes mellitus and severe peripheral vascular disease.

Unfractionated heparin may be used with ASA to control the thrombus. In patients with unstable angina, this combination may reduce mortality. Low-molecular weight heparins such as, enoxaparin is preferred for unstable angina or NSTEMI patients, unless percutaneous intervention or CABG is scheduled in the next 24 hours. Enoxaparin are not reversible, but unfractionated heparin has a shorter half life and may be emergently reversed with protamine sulfate.

With ECG findings of at least 1 mm ST-segment elevation in two or more contiguous leads, the patient requires intervention for STEMI. The preferred method is percutaneous coronary intervention (PCI), but distance to centers and time delays limit this practice to be administered to every patient. Fibrinolytics may be considered as an alternative to either angioplasty or stent placement. Fibrinolytics should be administered within 6–12 hours. If there is access to a cardiology consult, one should be obtained before administering. Patients should be selected carefully because fibrinolytics carry significant risk. Elderly patients over 75 years of age have been shown to have significant complications. Alteplase or tissue plasminogen activators (tPAs) have been shown to have good rate of reperfusion and minimal risk within 90 minutes given a front-loaded dose. Hemorrhage, especially intracranial bleeding, is the most feared complication. Clopidogrel in combination with ASA is considered for unstable angina (UA)/NSTEMI patients since it reduces the composite

risk of cardiovascular (CV) death, MI, or stroke. Glycoprotein IIb/IIIa may be used as an adjunct to angioplasty, medical stabilization of ACS, and in combination with low-dose fibrinolytics.

Right ventricular infarcts have special considerations. These types of infarcts are preload dependent to maintain cardiac output, therefore, diuretics and nitroglycerin should be avoided. Cardiac output may need to be maintained with volume infusion. Dobutamine should be used if an inotropic agent is needed. Nitroprusside or intra-aortic balloon counterpulsation is available in refractory cases.

ASSESSMENT

Tests

Electrocardiogram

- Persistent ST-segment elevation 2 mm in two contiguous limb leads
- ST-segment elevation 3 mm in two contiguous chest leads
- *New left bundle branch block pattern*: QRS 130 ms. V1: rS complex. V6: RsR'
- *Cardiac biomarkers*: Elevation of biomarkers such as troponin, creatine kinase muscle and brain (CK-MB) 24 hours post-onset of symptoms.

MANAGEMENT

Following measures should be taken as soon as possible (ASAP):

- Cardiac monitor
- O_2 therapy
- IV line should be established ASAP
- Chew aspirin
- Nitroglycerin
- Pain control by morphine
- Reperfusion strategy ASAP, fibrinolytic therapy or PCI
- *ER reperfusion*: restores coronary patency
- *Percutaneous coronary intervention*: best if promptly provided.
    - Short- and long-term benefit if done within 12 hours of symptom onset versus thrombolysis.
    - Less than 90 minutes from first medical encounter to PCI.
    - Transfer to facility capable of performing PCI if can be done within 90 minutes
- If PCI is not available, thrombolysis/fibrinolysis
    - Streptokinase or tPA
        - Restore perfusion to ischemic area
        - Lyse clot, reduce infarct size and improve survival

        - Most beneficial for anterior infarction
        - Early implementation is better
        - Greatest benefit recorded is for ST elevation or bundle branch block (BBB) with symptom onset within 12 hours
    - IV streptokinase and alteplase
    - *Rapid bolus injection*: Reteplase, tenecteplase
    - *tPA*: Most common agent

Contraindications of Streptokinase

- Prior use less than 12 months due to antibody persistence
- Consider thrombolytic as alternative to PCI in patient with STEMI with more than 1 mm ST elevation in two contiguous leads.
- New LBBB.

Absolute Contraindications

- Active bleeding, bleeding diathesis
- Significant closed head or facial trauma less than 3 months
- Suspected aortic dissection
- Prior intracranial hemorrhage
- Ischemic stroke within 3 months.

Relative Contraindications

- Active peptic ulcer
- Severely, poorly control HTN
- Ischemic stroke within 3 months
- Late presentation
- *Reperfusion with PCI or fibrinolysis*: Not recurrent if > 12 hours after symptoms onset, asymptomatic versus stable angina
    - CABG more appropriate.
    - Cardiogenic shock with mechanical repairing.

Adjuvant Therapy

- *Antiplatelet therapy*: Aspirin 300 mg. Low-dose followed on long-term basis.
    - Clopidogrel
        - 300–600 mg loading dose if PCI with stent is planned
        - 300 mg with aspirin if used for fibrinolysis
        - withhold if CABG is required acutely
        - 75 mg daily
        - 1 month after thrombolysis

        - 9–12 months after stent.
- *Antithrombin therapy*
    - *With PCI*: Unfractionated heparin (UFH) + Glycoprotein (GP) IIb/IIIa inhibitors
    - *With fibrinolysis*: Unfractionated heparin
    - Recommended dose of UFH is an initial bolus of 60 units/kg body weight (maximum 4,000 units) followed by an initial infusion of 12 units/kg/hr, maximum units 1,000/hr
    - Adjusted to attain activated partial thromboplastin time (aPTT) at 1.5–2 times the control value.
    - Enoxaparin in conjunction with fibrin-specific fibrinolytic agent can be used
    - If less than 75 years, no renal dysfunction
    - *GP IIb/IIIa inhibitors*: abciximab + Primary PCI with full-dose fibrinolytic therapy.

Cardiac Surgery

- Coronary Artery Bypass Surgery (CABG) is performed if PCI fails with pain or hemodynamic instability and coronary anatomy is suitable.
- Persistent/Recurrent ischemia refractory to medical therapy.

Differential Diagnosis

- Costochondritis
- Hiatal hernia
- GERD
- Peptic ulcer disease
- Gall bladder disease
- STEMI
- Aortic stenosis
- Myocarditis
- Pericarditis
- Pleuritis
- Dissecting aortic aneurysm
- Mitral valve prolapse
- CHF
- Pulmonary embolism
- Pulmonary HTN
- Pneumothorax
- *Diagnosis*: STEM

PLAN

- Discharge medications after ACS
- *Aspirin*: 75–325 mg PO daily
- *Clopidogrel*: 75 mg PO daily for 9–12 months after ACS if aspirin is contraindicated.
- *Beta blockers*: Metoprolol or carvedilol PO daily lifelong 50 mg PO daily.
- *Angiotensin converting enzyme inhibitors*: Used in ACS patient in CHF left ventricular (LV) dysfunction with ejection fraction (EF) less than 40%. Discharge if heart failure resolves.
- *Statin*: Initiate in hospital.
- *Nitrates*: Short-acting nitrates are used.
- *Warfarin*: Used only if high-risk thromboembolism is present because of atrial fibrillation, mural thrombus and CHF.
- *Address risk factors*: Hyperglycemia, HTN control, tobacco cessation, physical inactivity, alcohol.
- Exercise ECG testing:
- *Submaximal testing*: 4–7 days postinfarction, *Maximum*: 3–6 weeks postinfarction.
- Identify patient with recurrent ischemia, who needs angiogram to assess CABG
- *Myocardial perfusion imaging*
  - It assesses the residual ischemia extent.
  - It should be done before cardiac catheterization and angiography.

DISCUSSION

Acute Coronary Syndrome
These are of three types: (1) Unstable angina, (2) NSTEMI and (3) STEMI.

Causes

Coronary versus atherosclerotic obstruction, superimposed thrombotic occlusion.

Site

Infarction in areas of nonlimited blood flow at rest: Noncritical coronary stenosis.

Pathophysiology

- Periods of relative quiescence
- Interposed with episodes of rapid and abrupt plaque development and changes

- Plaque disruption and mural thrombosis
- *Rate-limiting mechanisms*: Acute thrombosis—resultant obstruction of coronary lumen.

Angiography

- Noncritical lesions account for majority of ACS
- "Red" thrombi, fibrin-rich for STEMI.

Duration

- Symptoms remain for more than 20 minutes and do not respond completely to nitroglycerin
- Symptoms may resolve completely after a few hours or persist for more than 24 hours.

Comorbidities

- Elderly, diabetes mellitus
- One-fifth MIs are "silent" and may not seek medical treatments.

Presentation

- Anxiety and pain
- Diaphoresis
- *Pulse rate*: Normal; *bradycardic*: inferior infarction, *tachycardic*: large infarction
- *BP*: Elevated
- *Cardiac activity*: Usually normal
- *Large infarction*: Ventricular failure, valve dysfunction
- *S4*: Stiffened ventricle
- *Mitral regurgitation*: Papillary muscle malfunction
- S2 paradoxically split at LV control time increases due to LBBB, weakened LV
- *Progression*
  - *Later*: Mild fever
  - Pericardial friction rub
  - Ventricular septal defect murmur due to septal rupture
  - Severe mitral regurgitation due to papillary muscle rupture.

PRACTICE PEARL AND PITFALL

Women are at a greater risk for misdiagnosis because they tend to have atypical symptoms and false negative stress tests. Consider any extreme

fatigue in a young woman an MI unless proved otherwise. Painless MIs are associated with higher mortality than painful MIs.

ADDITIONAL READING

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4. Coven DL. Acute Coronary Syndrome Medication. [online] Available from emedicine.medscape.com/article/1910735-medication#2. [Accessed June, 2012].

Case Study 18: Acute Coronary Syndrome—Non-ST -Segment Elevation Myocardial Infarction

CASE HISTORY

Patient is a 60-year-old Caucasian female who presents with chest pain for 1 hour. Patient states that "it feels like a heavy weight on my chest. It won't go away." Pain is constant, substernal/epigastric radiating to her neck, left shoulder and left arm. She has a past history of hypertension (HTN), diabetes, coronary artery disease (CAD), and stroke for which she takes acetylsalicylic acid (ASA), humalog, metformin, enalapril and atenolol. She has smoked two packs of cigarettes per day for the past 40 years and drinks a glass of wine daily. She used cocaine regularly in the past, but has been in remission for 30 years. Her vital signs are temperature of 36°C, blood pressure 95/65 mm Hg, pulse rate 110 beats/minute and respiratory rate 20 breaths/minute. On physical examination, skin is pale and cool. Cardiac examination reveals tachycardia with an S3. Bibasilar pulmonary rales are present on lung examination. Remainder of examination is unremarkable. Electrocardiogram (ECG) reveals dynamic ST-segment depression of 0.6 mm with a new T-wave inversion of 3 mm as well as sinus tachycardia. Cardiac markers are negative. Patient has a presumed non-ST-segment elevation myocardial infarction (NSTEMI) and medical management is initiated (Fig. 1). Patient is admitted for further evaluation.

DISCUSSION

Differential diagnosis includes costochondritis, hiatal hernia, gastroesophageal reflux disease (GERD), peptic ulcer disease, gall bladder disease, aortic stenosis, mitral valve prolapse, myocarditis, pericarditis, dissecting aortic aneurysm, pleuritis, congestive heart failure (CHF), pulmonary embolism, pulmonary hypertension and pneumothorax.

Initially unstable angina (UA) and NSTEMI have similar presentations. Unstable angina or ischemic equivalent has one of the three features (1) occurs at rest or with minimal exertion, lasting more than 10 minutes, (2) severe and of new onset, prior within 4–6 weeks, (3) occurs with crescendo pattern, more severe, prolonged and frequent than before. NSTEMI has the clinical features of UA, but develops evidence of myocardial necrosis with elevated cardiac biomarkers, creatine kinase muscle and brain (CK-MB), troponin and over time.

A list of acute coronary syndromes as they increase in severity includes: UA occurring without serologic evidence of myocardial necrosis; new-onset angina, severe and lasting longer than 2 months; increasing (crescendo) angina which is gaining in severity, length or frequency. Angina pectoris usually occurs at rest, prolonged pain for greater than 20 minutes and within 1 week of presentation. Postinfarction angina occurring 2 weeks after an acute myocardial infarction (MI).[1] Acute MI is next which includes the following: Non-ST-elevation (Non-Q-wave) MI, and a transient thrombotic occlusion with early spontaneous reperfusion. Also, the ST-elevation (Q-wave) MI, a total thrombotic occlusion which is associated with a larger infarct size and higher in-hospital mortality rate. As in all cases, earlier the reperfusion therapy begins the better is the probability of preventing sudden death and also decreases infarct size. "Time is Muscle". Sudden ischemic cardiac death, secondary to malignant ventricular tachyarrhythmias, is the most severe type of acute coronary syndrome.

All patients with suspected acute coronary syndrome should be administered Aspirin 325 mg. NSTEMI management differs from STEMI. Patients with a suspected NSTEMI should initially be medically managed. They are still candidates for percutaneous coronary intervention (PCI), but likely during hospital admission rather than immediately. Patients should be given anticoagulants. Generally, patients are administered plavix and lovenox unless surgical intervention is planned within 24 hours. Nitrates and/or morphine may be used for pain control. Patients should also be given an angiotensin-converting-enzyme (ACE) inhibitor and beta blocker in the first 24 hours.

The cause of acute coronary syndrome regardless is a reduction in oxygen (O_2) supply and increase in myocardial O_2 demand. The cause may be superimposed on an atherosclerotic coronary plaque with varying degrees of obstruction. There are several pathophysiologic possibilities. First, there may be plaque rupture/erosion with superimposed nonocclusive thrombus causing a NSTEMI downstream embolization of platelet aggregation/atherosclerotic debris. Second, there may be dynamic obstruction causing coronary spasm and Prinzmetal's variant angina. Third, there may be a progressive mechanical obstruction causing rapidly advancing coronary atherosclerosis/restenosis after PCI. Finally, there may be secondary UA relative to increased myocardial O_2 demand or decreased supply.

The common findings on coronary angiography are 5% left main stenosis, 15% three-vessel (CAD), 30% two-vessel disease, 40% single-vessel disease, and 10 without critical coronary stenosis. Prinzmetal's variant *culprit lesion* may show an eccentric stenosis with

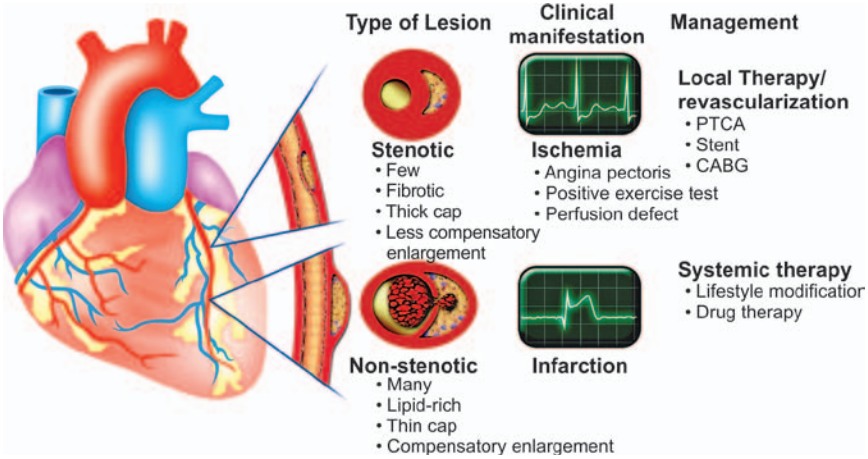

Fig. 1: Clincal Management of NSTEMI

scalloped/overhanging edges and narrow neck *white* thrombi platelets with rich multiple plaque vulnerable to disruption.

High-risk features for patients with suspected UA/NSTEMI are repetitive or prolonged chest pain lasting more than 10 minutes, elevated cardiac biomarkers, persistent (ECG) changes, hemodynamic instability with systolic blood pressure less than 90 mm Hg, sustained ventricular tachycardia, syncope, left ventricular ejection fraction (LVEF) below normal 40%, prior percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass graft (CABG), diabetes and chronic kidney disease.

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Case Study 19: Unstable Angina

CASE HISTORY

Patient is a 63-year-old male with past medical history (PMH) of hypertension and hyperlipidemia who presents with worsening chest pain. He has had chest pain in the past while working in the yard, but in the past it always went away after a few minutes when he sat down to rest. This morning he was mowing the lawn and started experiencing a pressure-like pain in his chest that radiated to his left arm. He sat down to rest, but the pain did not go away so his wife called for an ambulance. He was given two nitroglycerin tablets en route which have not alleviated the pain. He also received four baby aspirin while en route. Pain has now lasted approximately 40 minutes. His vital signs on arrival are temperature of 38.6°C, blood pressure 160/90 mm Hg, pulse rate 105 beats/minute, respiratory rate 20 breaths/minute and O_2 saturation of 96% on 2 L nasal cannula. Intravenous (IV) lines are started and patient is placed on the cardiac monitor. Electrocardiogram (ECG) and chest X-ray (CXR) are ordered which are shown below (Figs 1 and 2). Patient is given morphine which temporarily relieves the pain, but it returns again after approximately 20 minutes. Serial ECGs and cardiac enzymes are ordered. Pain is thought to be cardiac in origin with possible unstable angina.[1] How should this patient be managed?

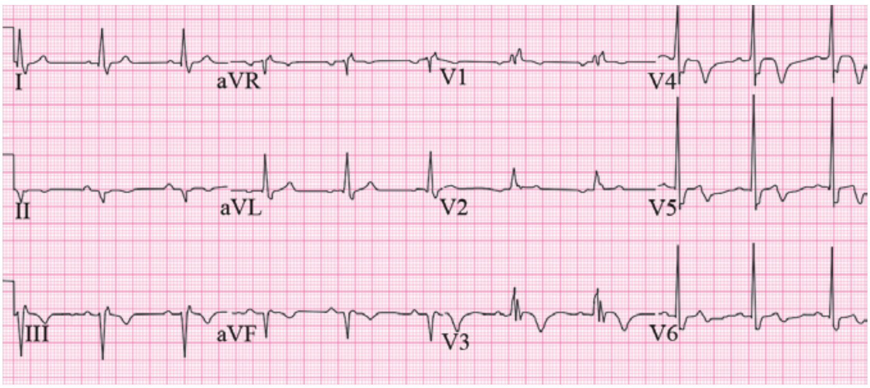


Fig. 1: ECG

Source: EMT Emergency Medicine Tutorials. [online] Available from http://www.emergency-medicine-tutorials.org/Home/medical-3/cardiovascular/ecgs-1/wellen-s-syndrome-sign-warning. [Accessed June, 2012].

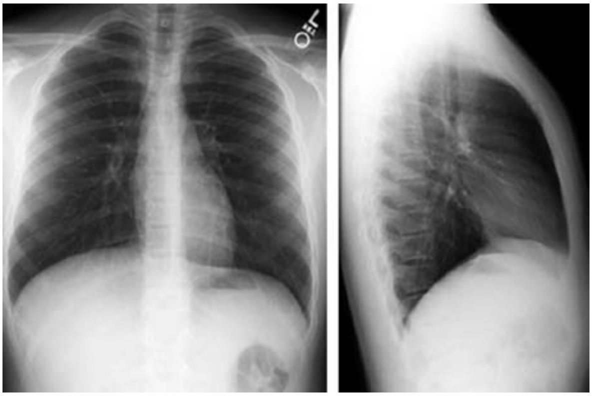


Fig. 2: Chest X-ray (CXR) of a 63-year-old male patient

DISCUSSION

Please see approach to chest pain discussion in Case Study 17: Acute Coronary Syndrome—ST-Segment evaluation in myocardial infarction.

Unstable angina is a type of acute coronary syndrome in which the patient does not experience a release of cardiac enzymes. Causes may include unstable or disrupted atherosclerotic plaques. Other factors may include supply-demand mismatch, plaque disruption or rupture, thrombosis, vasoconstriction, or cyclical flow.

Overall, the number of patients with coronary unstable angina is rising. The trends are slightly different depending on age and sex. Women tend to be older and have higher rate of hypertension, diabetes, congestive heart failure (CHF), and family history of coronary artery disease (CAD) when compared to men, whereas men tend to have higher rate of previous myocardial infarction (MI), more positive cardiac enzymes and higher rate of catheterization. Overall patients tend to range in age from 23 years to 100 years with a median age of 62 years.

Several factors have been shown to suggest a poor outcome including ongoing CHF, poor left ventricular ejection fraction (LVEF), hemodynamic instability, recurrent angina, new or worsening mitral regurgitation and sustained ventricular tachycardia.

A rapid but thorough history should be obtained with a focus on the symptoms and coronary risk factors. ECG should be rapidly obtained. Physical examination should include assessment of vital signs and consideration of other illnesses including aortic dissection, leaking or ruptured thoracic aneurysm, pericarditis with tamponade, pulmonary embolism and pneumothorax.

Unstable angina is generally more intense than stable angina. Pain is ischemic in nature with either a sensation of heaviness, tightness, aching,

Table 1: Braunwald classification of unstable angina

Characteristic	*Class/Category*	*Details*
Severity	I	Symptoms with exertion
	II	Subacute symptoms at rest (2–30 days prior)
	III	Acute symptoms at rest (within prior 48 hours)
Clinical precipitating factor	A	Secondary
	B	Primary
	C	Postinfarction
Therapy during symptoms	1	No treatment
	2	Usual angina therapy
	3	Maximal therapy

Note: "Patients in Class I have new or accelerated exertional angina, whereas those in Class II have subacute (> 48 hours since last pain) or Class III acute (< 48 hours since last pain) rest angina. The clinical circumstances associated with unstable angina are categorized as (A) secondary (anemia, fever and hypoxia), (B) primary, or (C) postinfarction (< 2 weeks after infarction). Intensity of antianginal therapy is subclassified as (1) no treatment, (2) usual oral therapy, and (3) intense therapy, such as IV nitroglycerin."

Source: Tan WA. (2011) Unstable Angina. [online] Available from emedicine.medscape.com/article/159383-overview#showall. [Accessed June, 2012].

fullness, or burning in the chest, epigastrium and/or arm or forearm. Associated symptoms might be dyspnea, generalized fatigue, diaphoresis, nausea and vomiting, flu-like symptoms, lightheadedness or abdominal pain (Table 1).

The thrombolysis in myocardial infarction (TIMI) risk score may be used as a clinical prediction of severity. The risk increases greatly after a score of 3. Patients with scores of 3–7 should be considered for anticoagulation. Each of the following represents one point:

- Aged 65 years or older
- Use of aspirin in the last 7 days
- Known coronary stenosis of 50% or greater
- Elevated serum cardiac markers
- At least three risk factors for CAD (diabetes, smoking, family history of CAD, hypertension, hypercholesterolemia)
- Severe anginal symptoms (Two or more anginal events in the last 24 hours)
- ST deviation on ECG

Workup in the first 24 hours should include serial cardiac biomarkers, hemoglobin, chemistry and lipid panel. Stress testing should not be performed in the acute phases of unstable angina (Fig. 3).

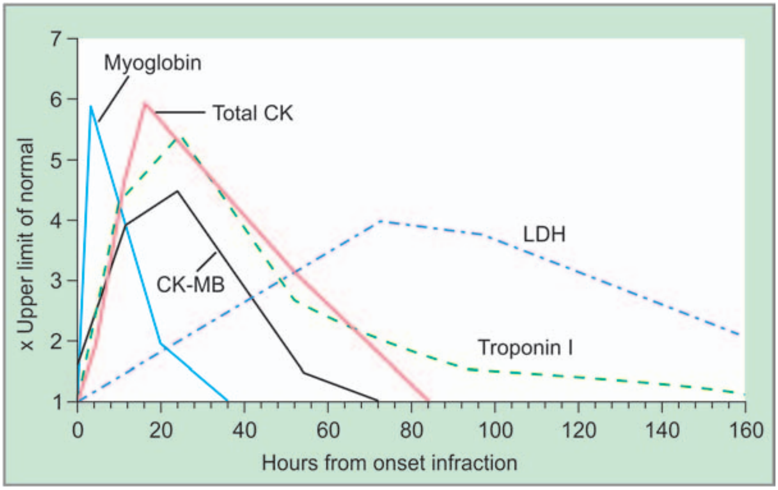
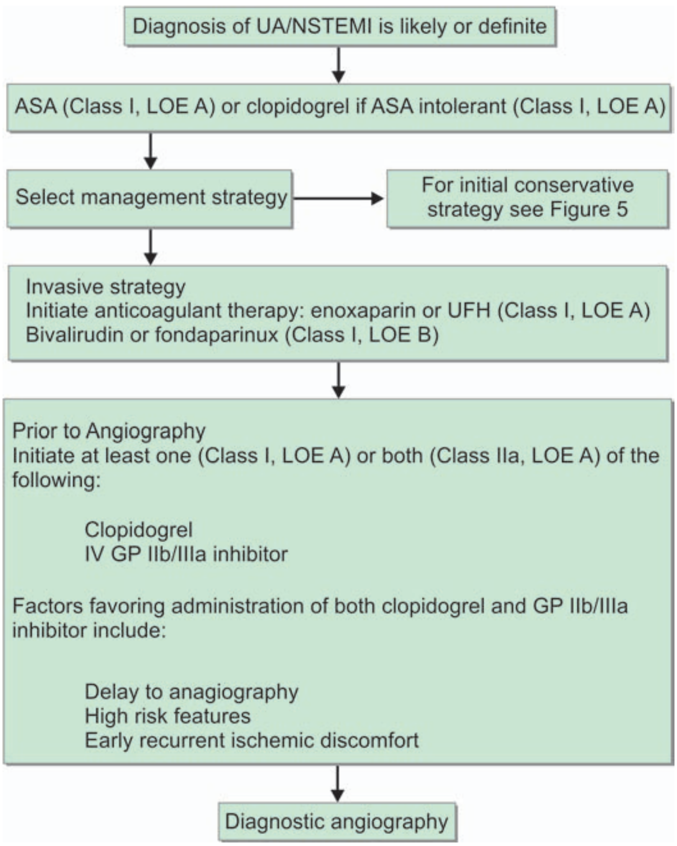


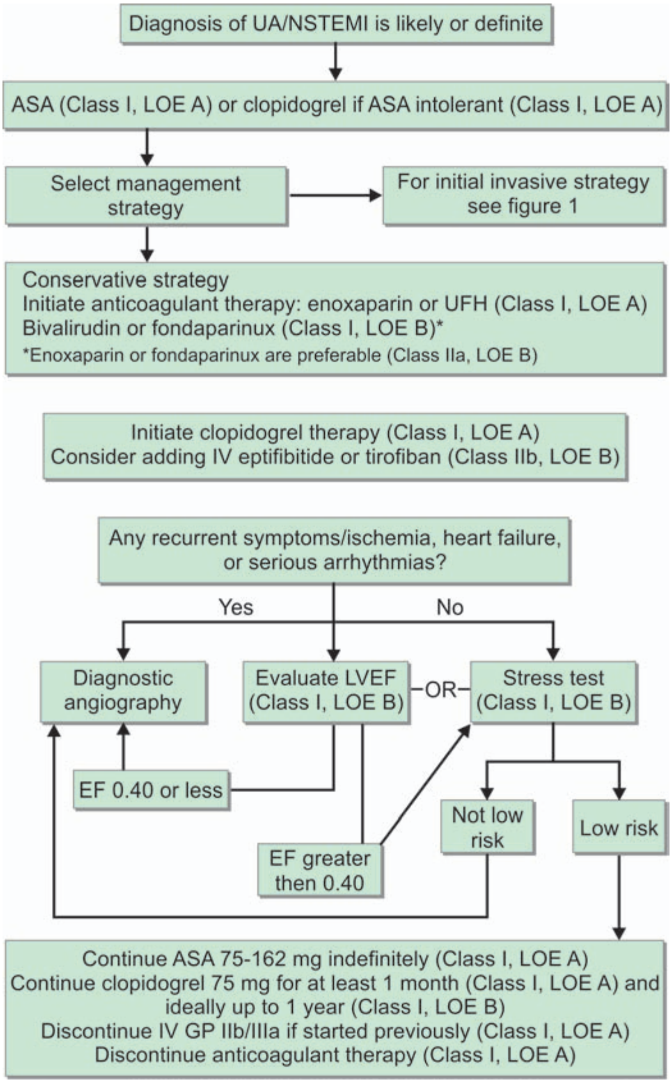
Fig. 3: Time course of elevations of serum markers after acute myocardial infarction
Source: Tan WA. (2011) Unstable Angina. [online] Available from emedicine.medscape.com/article/159383-overview#showall. [Accessed June, 2012].

Flow chart 1: Algorithm for initial invasive strategy



Source: Adapted from 2007 ACC/AHA UA/NSTEMI Guidelines

Flow chart 2: Algorithm for initial conservative strategy



Source: Adapted from 2007 ACC/AHA UA/NSTEMI Guidelines

The following algorithms shown in Flow charts 1 and 2 are helpful in determining management.

REFERENCE

1. Tan WA. (2011) Unstable Angina. [online] Available from emedicine.medscape.com/article/159383-overview. [Accessed June, 2012].

Case Study 20: Abdominal Aortic Aneurysm

CASE HISTORY

Patient is a 70-year-old male with a 50-year smoking history who presents to the emergency department (ED) after passing out while sitting on the couch watching TV. Right before passing out, he complained to his daughter that he had a sudden onset, intense abdominal pain that radiated to his back. Patient's daughter denies recent trauma. However, he recently experienced some stomach discomfort and has had multiple episodes of nausea and vomiting in the last couple of days. The daughter denies that the patient had chest pain, shortness of breath (SOB), fever, diarrhea or constipation. He has a 20-year history of hypertension, coronary artery disease (CAD), and dyslipidemia as well as a 10-year history of non-insulin-dependent diabetes mellitus (NIDDM). His vital signs on admission are insignificant except for a blood pressure (BP) of 140/90 mm Hg. On physical examination, patient is now awake and alert, but still appears drowsy. Heart examination reveals systolic murmur 2/6 near mitral area with S1, S2 and S4. Respiratory examination reveals wheezing. Abdominal examination reveals pulsatile abdominal mass.

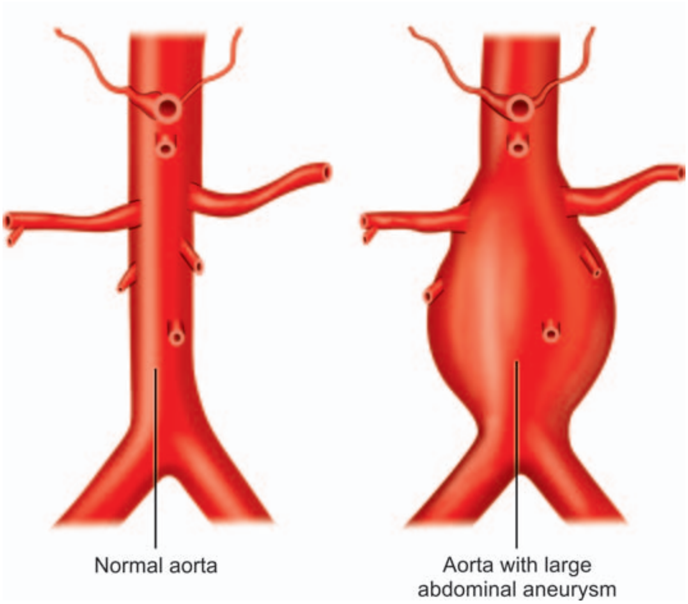


Fig. 1: Abdominal Aortic Aneurysm

Source: Available online from http://www.drfansler.com/images/abdominal/aortic/aneurysm-320.jpg

Electrocardiogram (ECG) shows left ventricular hypertrophy. Abdominal ultrasound is done immediately and show enlarged aorta without free peritoneal fluid (see Fig. 7B).

DISCUSSION

The immediate concern with this elderly patient with a history of smoking is a ruptured abdominal aortic aneurysm (AAA) versus a myocardial infarction.[1] These must be ruled out immediately. The history of syncope is concerning, but it is reassuring that the patient is not significantly hypotensive. Although, given his history of hypertension, this BP may be relatively hypotensive compared to his baseline. A broader differential includes myocardial infarction, appendicitis, cholelithisasis, diverticular disease, gastritis and peptic ulcer disease, large bowel obstruction, pancreatitis, small-bowel obstruction, gastrointestinal (GI) bleeding, ischemic bowel, perforated ulcer, *urinary tract infection* (UTI), nephrolithiasis, pyelonephritis and musculoskeletal pain. Please (see Figure 1) to understand the anatomy of abdominal aortic aneurysm.

A good initial step to check for an enlarged abdominal aorta is by bedside ultrasound (Fig. 2). Limitations of the study are few but include inability to detect leakage, rupture, branch artery involvement and suprarenal involvement. In addition, the ability to image the aorta is reduced in the presence of bowel gas or obesity. Visualization may be further limited if patients are non-fasting. Other

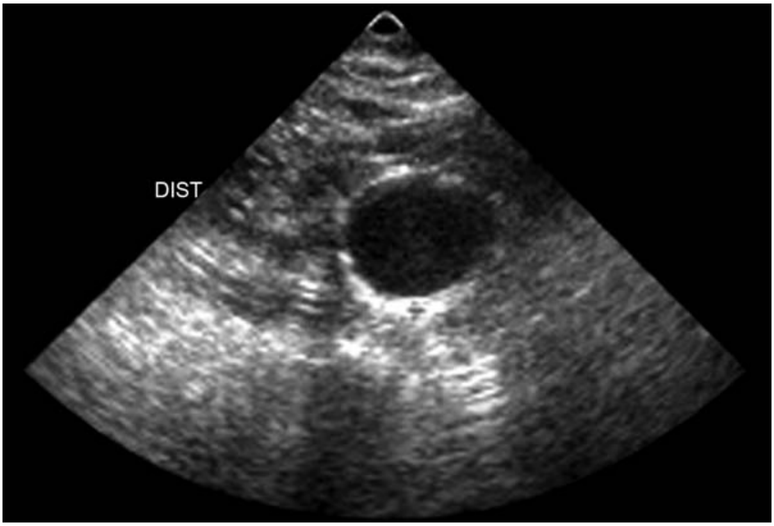


Fig. 2: A bedside ultrasound of an enlarged abdominal aorta
Courtesy of Dr Beatrice Hoffman at John Hopkins University www.sonoguide.com

early studies should include ECG, chest X-ray (CXR), complete blood count (CBC), *basic metabolic panel* (BMP), and type and screen.

Other causes of abdominal pain may be investigated using abdominal radiography (Fig. 3).

A secondary study to evaluate and detect AAA is an abdominal CT scan with a sensitivity of almost 100% (Fig. 4). CT may visualize the entire aorta as well as branches and the retroperitoneum. The limitations are time, cost and exposure to radiation.

Magnetic resonance imaging (MRI) is similar in benefits to CT scan. However, MRI is even more limited in access and is more expensive (Fig. 5).

Finally, angiography is the gold standard, but widespread use is limited by its invasiveness, cost, lack of operator availability, time involved and risk of complications such as bleeding, perforation or embolism (Fig. 6). Therefore, ultrasound and CT are the most highly recommended studies for evaluating abdominal aneurysms.

Treatment initially depends on the hemodynamic stability of the patient. Unstable patients require immediate surgical intervention. Mortality rate for ruptured aneurysms is extremely high even with intervention is initiated in a timely fashion. BP control is important as

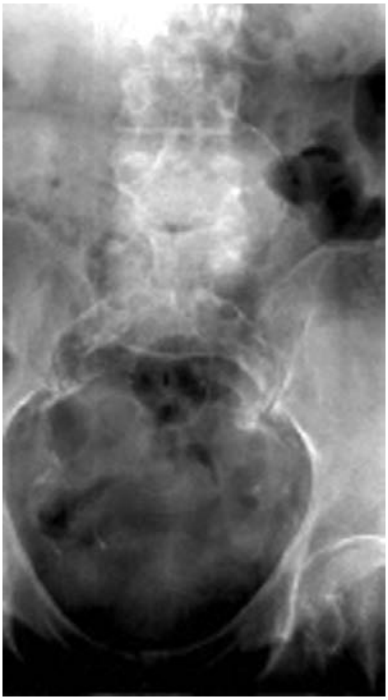


Fig. 3: Radiograph shows calcification of the abdominal aorta. The left wall is clearly depicted and appears aneurysmal; however, the right wall overlies the spine

Fig. 4: CT demonstrates an abdominal aortic aneurysm (AAA). The aneurysm was noted during workup for back pain, and CT was ordered after the AAA was identified on radiographs. No evidence of rupture is seen

Fig. 5: MRI scan of a 77-year-old man with leg pain believed to be secondary to degenerative disk disease. During evaluation, an abdominal aortic aneurysm was discovered

Fig. 6: Angiography is used to diagnose the renal area. In this instance, an endoleak represented continued pressurization of the sac

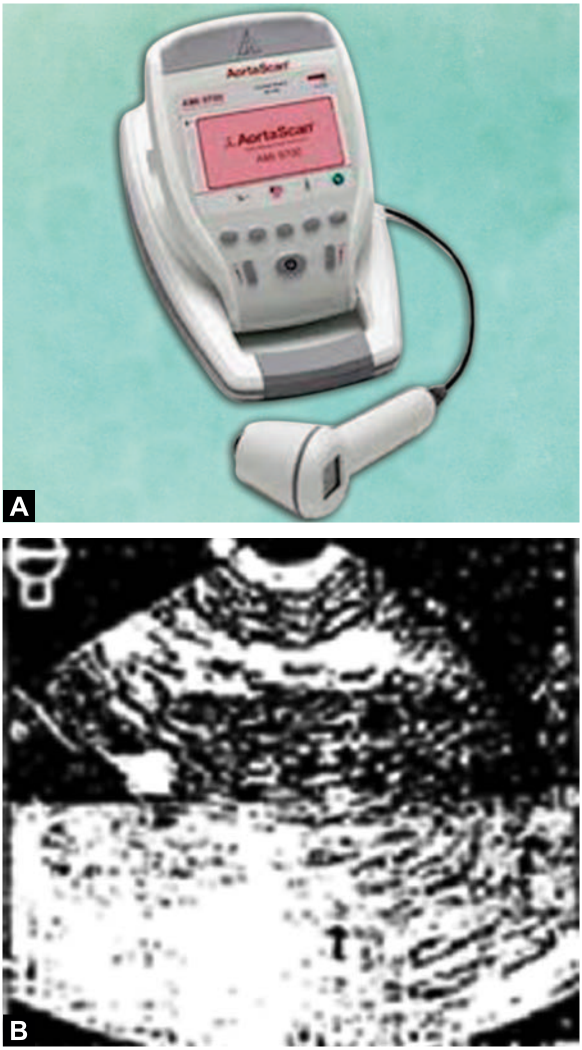
not to exacerbate the bleed. A short-acting beta blocker, such as esmolol is ideal. Stable but symptomatic patients should be admitted especially when comorbid conditions are present such as chronic obstructive pulmonary disease (COPD), CAD and congestive heart failure (CHF).

Stable patients can be monitored on a yearly basis. If the aneurysm grows to greater than 5.5 cm or is growing at a rate of 1 cm per year then surgical intervention is recommended. This is the generally accepted point where risk of rupture outweighs the risk of surgery. Long-term medical management should include smoking cessation, and aggressive BP control with beta blockers.

Several repair mechanisms may be used and are to be decided by the vascular surgeon and the patient. Open repair and endovascular repair are the main differences. Endovascular repair has lower morbidity and mortality, but is limited to certain types of aneurysms. Historical Case: Albert Einstein died of severe bleeding from ruptured aortic aneursym.

AORTA SCAN

This has been represented with a portable 3D ultrasound device which helps the physicians to measure the abdominal aortic diameter quickly and accurately (Figs 7A and B).



Figs 7A and B: Aorta Scan® AMI 9700. Available online from http://www.verathon.com/Products/AortaScan/AMI9700.aspx. [Accessed June, 2012]
Source: With permission from Verathon

REFERENCE

1. O'Connor RE. (2011) Emergent Management of Abdominal Aortic Aneurysm Rupture. [online] Available online from emedicine.medscape.com/article/756735. [Accessed June, 2012].

Case Study 21: Hemorrhagic Shock

"None of our patients should code in the CT scanner"

—Badar M Zaheer

CASE HISTORY

After a heated argument with his wife and two daughters, a 45-year-old male began drinking heavily. He punched through a window and cut his wrists with the glass "hoping to die". The patient then drove himself to the emergency department (ED) and stood in the parking lot drinking hard liquor, while bleeding profusely. Initial attempts to bring him into the ED for treatment were met with physical resistance. The patient agreed to come inside for treatment but he continued to curse and yell at the staff the entire time. He continued to become angry and more unintelligible. Once the patient's arms were secured, normal saline was started and the bleeding at his wrists was stopped. The patient became increasingly lethargic and O+ blood was ordered.

There are a number of conditions we should be looking for in the setting of shock. A bedside ultrasound scan can help you narrow down your differential: pericardial tamponade, pulmonary embolism (PE), myocardial infarction (MI), hypovolemia, aortic dissection and rupture of abdominal aortic aneurysm (AAA) are some at the top of the list. Since there is no room to show all of these here, you may want to check out the free ultrasound image library at www.erpocketbooks.com as you read on.

With cardiomegaly on the chest X-ray, especially if you don't know that it is chronic, your first consideration should be cardiac tamponade from a large pericardial effusion. A bedside echo should be able to rule this out quite easily. On a quick look, your patient does not have any signs of an effusion, so you move on. Given the heart murmur, your next consideration should probably be cardiogenic shock, and if this were the case you would expect to see a dilated, hypokinetic left ventricle (LV). However, the LV is actually small and it is the right ventricle (RV) that appears enlarged. This should make you suspect a PE, however this was eventually ruled out with a pulmonary angiogram. As it turned out, this patient was eventually diagnosed with acute pulmonary hypertension, possibly due to a combination or cirrhosis and methamphetamine abuse (see above for facts on pulmonary hypertension).

Other conditions that the ultrasound machine could be helpful with include hypovolemic shock, where you would expect to see a small rapidly beating heart and a shrunken or flat inferior vena cava (IVC). If you look carefully in the right patient, it is possible to pick up an intimal

flap from an acute aortic dissection. Imaging the belly for an AAA or free fluid from a ruptured spleen or hemorrhagic pancreatitis might also be helpful in narrowing down the cause of shock in a sick patient. In the setting of a new murmur, valvular pathology or an acute MI should also be considerations. Imaging for these conditions, however, is beyond the scope of most emergency physicians, so when you are worried about these a formal echocardiogram is desirable.

PEARLS AND PITFALLS FOR ULTRASOUND OF HYPOTENSIVE PATIENTS

- Start with the heart. Parasternal images tend to give the most information, but the subxiphoid view may be easier in some patients. Look for effusions, chamber dilation and wall motion abnormalities (advanced).
- Use the curvilinear probe or the phased array transducer. Start in the fourth intercostal space and position the probe between the ribs. If you do not get a good image, try moving up or down one rib space and redirecting the angle of your probe. Remember to direct the beams of your probe through the long axis of the heart. If you need to decrease the distance between the patient's heart and the ultrasound beams, have the patient rolled over onto his left side and bring his heart closer to his chest wall.
- Take the time to adjust your depth and gain to maximize image quality. Make sure you have enough depth to look behind the heart for a pericardial effusion. You should always try to see the cross-sectional descending aorta coursing behind the LV.
- In the setting of hypotension, pericardial fluid, especially when accompanied by right ventricular collapse, may signify tamponade, a condition which is best treated by immediate pericardiocentesis (see Fig. 3). Pleural effusions may mimic pericardial effusions in certain instances. If you are unsure, find the retrocardiac aorta. If the fluid is between the heart and aorta, it is pericardial. If it is on the opposite side of the aorta from the heart, it is pleural.
- A hyperdynamic rapidly beating heart usually signifies hypovolemia. Look at the IVC to assess if the patient is indeed intravascularly depleted. Many studies have correlated IVC diameter to Swan Ganz RA pressures. If the heart is hyperdynamic and the IVC is compressed, the aggressive administration of fluids or blood products may be life-saving. Consider a search for the source of bleeding. Look for melena, hemoperitoneum or an aortic aneurysm.

- A dilated RV should raise your suspicion for a massive PE. In such cases, tissue plasminogen activator (tPA) should be considered. Remember that congestive heart failure (CHF) or pulmonary hypertension can also cause a dilated RV, although these chronic conditions will show a dilated RV with hypertrophy of the RV walls. A dilated RV with thin walls prompts consideration of the diagnosis of an acute massive PE.
- Scanning further south may also help to find the cause. Image the abdomen for evidence of retroperitoneal bleeding from a ruptured AAA, or intraperitoneal bleeding from occult trauma, or ruptured ectopic pregnancy or spleen. Ultrasound of the legs may show deep vein thrombosis (DVT).
- Ultrasound may be useful for more than just establishing a diagnosis. It can also help with procedures such as central line placement and pericardiocentesis during the resuscitation.
- Consider the concentrated overview of resuscitative efforts (CORE) scan in all undifferentiated hypotensive patients. For more information, visit apps.acep.org/WorkArea/DownloadAsset.aspx?id=42470

Brady Pregerson manages a free online EM ultrasound image library and is the author of the Tarascon Emergency Department Quick Reference Guide. For more information, visit EMResource.org.

Teresa S Wu is the Associate Residency Director and Director of Ultrasound and Simulation Based Training for the Maricopa EM Program in Phoenix.

Physical Examination

- *Vital signs*: *BP*: could not be measured, *Heart rate*: 163 beats/minute, *Respiratory rate*: 35 breaths/minute, *Body temperature*: 95.6°F.
- *Skin/mucus membranes*: Pale, Cold, Clammy sweat
- *Cardiac activity*: Tachycardic.

Differential Diagnoses

It includes shock (cardiogenic, toxic, hemorrhagic, obstructive shock, distributive shock and neurogenic shock).

Hypovolemic Shock

- *Loss of blood*:
  - *External hemorrhage*: Trauma or GI bleed
  - *Internal hemorrhage*: Hematoma, hemothorax, hemoperitoneum
- *Loss of plasma*: Burns, exfoliative dermatitis
- *Loss of fluid and electrolytes*:

- *External*: Vomiting, diarrhea, excessive sweating, hyperosmolar states *diabetic ketoacidosis* (DKA)
- *Internal (Third spacing)*: Pancreatitis, ascites and bowel obstruction.

Cardiogenic Shock

- *Dysrhythmia*: Tachyarrhythmia, bradyarrhythmia
- *Pump failure*: MI, cardiomyopathy
- *Acute valvular dysfunction*: Regurgitant lesions more common
- Rupture of ventricular septum or free ventricular wall.

Obstructive Shock

- Tension pneumothorax
- *Pericardial disease*: Tamponade, constriction
- *Pulmonary vasculature disease*: PE, pulmonary hypertension
- *Cardiac tumor*: Atrial myxoma
- Left atrial mural thrombus
- *Obstructive valvular disease*: Aortic or mitral stenosis.

Distributive Shock

Septic shock, anaphylactic shock, neurogenic shock, vasodilator drugs, acute adrenal insufficiency

DIAGNOSIS

Considering the patient's obvious signs of trauma and excessive binge drinking, a hypovolemic and hemorrhagic shock is the most likely diagnosis. Refer to Table 1 for hemorrhagic shock according to the amount of blood loss.

Work Up for Shock

Identifying Source of Bleeding

External bleeding (lacerations), pleural cavity (hemothorax), peritoneal cavity (bleeding from intra-abdominal injuries), pelvic girdle (pelvic fracture) and soft-tissue compartments (long-bone fractures)

Imaging

Computerized tomography can be used for unclear bleeding sources in head, abdomen and pelvis. Plain film can be used for extremities to show potential fractures (Fig. 1).

Fig. 1: CT scan shows hemorrhage into left retroperitoneum (arrow) and along illiopsoas compartment

Laboratory

During an acute hemorrhage, there is not much use for a complete blood count (CBC). The hemoglobin and hematocrit results will be normal initially because the body is losing equal normotonic fluid. Decrease in these results will show up after crystalloid fluid replacement has been initiated and the red blood cell (RBC) is essentially diluted.

Table 1: Clinical diagnosis of hemorrhagic shock using—advanced trauma life support (ATLS) classification of hypovolemic/hemorrhagic shock

Class	*Blood loss (mL)*	*Pulse*	*Blood pressure*	*Capillary blanch test**	*Respiratory rate (Breaths/ minute*	*Urine output (mL/hr)*	*Psychology*
Class I	750 (15%)	<100	Normal with normal or increased pulse pressure	Normal	14–20	> 30	Slightly anxious
Class II	750–1,500 (15–30%)	>100	Normal with decreased pulse pressure	Positive	20–30	20–30	Mildly anxious

Contd...

Contd...

Class	*Blood loss (mL)*	*Pulse*	*Blood pressure*	*Capillary blanch test**	*Respiratory rate (Breaths/ minute*	*Urine output (mL/hr)*	*Psychology*
Class III	1,500–2,000 (30–40%)	>120	Decreased with decreased pulse pressure	Positive	30–40	5–15	Anxious and co fused
Class IV	> 2,000 (>40%)	>140	Decreased with decreased pulse pressure	Positive	> 35	Negligible	Lethargic

MANAGEMENT

Fluid Loss

Initial Step

Isotonic crystalloid fluid (isotonic saline or lactated ringer solution) is given as the initial fluid. For each liter given, 300 mL stays in intravascular space meaning 3 mL of fluid = 1 mL of blood loss.

- Colloid (albumin, hetastarch, dextran) versus crystalloid—no clinical difference

Blood Transfusion

If 2–3 L of crystalloid solution is not enough to increase the patient's saturation and cardiovascular collapse is imminent, blood transfusion is the next step. Ideally, cross-typed blood is the best option. However, in an emergency setting, O– blood is given to women and O+ blood is given to men.

Hemorrhage

- Pressure, tourniquet, dressing and tamponade can be tried for external wounds.
- Surgical consult needed for intra-abdominal injuries.
- Interventional radiology-guided embolization can be used for advanced pelvic injuries.

ADDITIONAL READING

1. *Hemorrhagic Shock* visit www.ncbi.nlm.nih.gov/pmc/articles/PMC 1065003
2. Beatrice Hoffman MD, PhD, RDMS, John Hopkins http://www.sonoguide.com/introduction.html

Case Study 22: Pericarditis

CASE HISTORY

The patient is a 45-year-old male who presents with acute sharp retrosternal pain that radiates to the shoulder. The pain is worse with inspiration and better when leaning forward. The patient has been experiencing a racing heart. He has been having a fever for the last 2–3 days. The fever has stayed consistent and his breathing has quickened. He noticed that his breathing has become more difficult. He says that the pain has been constant for the last 6 hours. He has a history of gastroesophageal reflux disease (GERD) and esophageal spasm. Patient says that he has felt pain similar to this before but it would go away within a few minutes.

PHYSICAL EXAMINATION

Cardiovascular System

Tachycardic, regular, pericardial friction rub heard [coarse, high pitched, better in expiration at lower left sternal border (LLSB) with patient leaning forward (specifically, not sensitive)], new S3 present.

Respiratory System

Patient develops hypotension, elevated *jugular venous pressure* (JVP), and blood pressure falls 10 mm Hg with inspiration.

Laboratory Tests

- Complete blood count (CBC) of 12,000/μL with no shift
- Erythrocyte sedimentation rate (ESR) elevated
- Elevated C-reactive protein (CRP)
- Elevated cardiac enzymes include elevated creatine kinase-muscle and brain (CK-MB), and troponins

  Note: Increased troponins at young age and normal male gender.
- Electrocardiogram (ECG) shows ST-segment elevation that does not come back down to baseline.

DIAGNOSIS OF PERICARDITIS

Differential Diagnoses

- *Cardiovascular system*: Aortic dissection, aortic stenosis, coronary artery vasospasm, myocardial infarction

- *Respiratory system*: Pulmonary embolism
- *Gastrointestinal system*: Esophageal rupture, esophageal spasm, esophagitis, gastritis, gastroesophageal reflux disease and peptic ulcer disease.

Physical Finding

- Increased heart rate out of proportion to fever
- S1, S4 faint on auscultation
- Mitral regurgitation murmur
- Pericardial friction rub
- The most frequent presentation is dyspnea (72% of all patients) followed by chest pain (32%) and then arrhythmias (18%).
- Signs of right heart failure (hepatomegaly, edema, distention in neck veins and loud S3)
- Fever, malaise and arthralgias. Difficulty in breathing may be the most common presentation in children.
- Sudden cardiac death.

WORKUP

- *Medical history*: The clinical presentation of myocarditis is nonspecific and can consist of fatigue, palpitations, dyspnea, precordial discomfort and myalgias.
- Diagnostic workup includes chest X-ray (CXR) examination, (ECG), laboratory evaluation, echocardiogram, cardiac catheterization and endomyocardial biopsy (in selected patients on the basis of the likelihood of finding specific treatable disorders such as giant cell myocarditis).

Laboratory Studies

- Elevated cardiac troponin T is suggestive of myocarditis in patients with clinically suspected myocarditis. Troponin I specificity is 89%; sensitivity is 34%. A normal level does not rule out the diagnosis.
- Increased creatine kinase (with elevated MB fraction, lactate dehydrogenase), and aspartate aminotransferase from myocardial necrosis.
- Increased ESR (nonspecific but may be of value in following the progress of the disease and the response to therapy).
- Increased white blood cell count (increased eosinophils in case of if parasitic infection).
- Viral titers (acute and convalescent)
- Cold agglutinin titer, antistreptolysin O titer

- Blood cultures
- Lyme disease antibody titer.

Imaging

- *Chest radiograph*: Enlargement of cardiac silhouette.
- *Electrocardiogram*: Sinus tachycardia with nonspecific ST-T-wave changes; intraventricular conduction defects and bundle branch block may be present:
  - Lyme disease and diphtheria cause all degrees of heart block.
  - Changes of acute myocardial infarction can occur with focal necrosis.
- *Echocardiogram*:
  - Dilated and hypokinetic chambers
  - Segmental wall motion abnormalities
- *Cardiac catheterization and angiography*:
  - To rule out coronary artery disease and valvular disease.
- A right ventricular endomyocardial biopsy can confirm the diagnosis, although a negative biopsy result does not exclude myocarditis. Recent studies have shown that myocardial biopsy may be unnecessary because immunosuppression therapy based on biopsy results is generally ineffective.

PERICARDITIS STAGING (FIGS 1 TO 5)

Stage 1 = Diffuse ST elevation, PR-Segment depression
Stage 2 = Normalization of ST and PR segments
Stage 3 = Widespread T-wave inversion
Stage 4 = Normalization of T-waves, persistent inversion if chronic

- ECG may show electrical alternans with tamponade.
- CXR shows enlarged cardiac silhouette with large pericardial effusion of at least 200 mL (see Fig. 2).
- Evaluating the presence of pericardial effusion tamponade of pericardial disease by transthoracic echo (see Fig. 5).
- Computed tomography/magnetic resonance imaging (CT/MRI) is done if initial workup is inconclusive.

DIAGNOSIS

- Pericardiocentesis (see Fig. 3) is indicated for cardiac tamponade, high suspicion of tuberculous, purulent, or neoplastic pericarditis
  - Purulence with neutrophil shows bacterial pericarditis
  - Purulence with lymphocytes shows neoplastic pericarditis

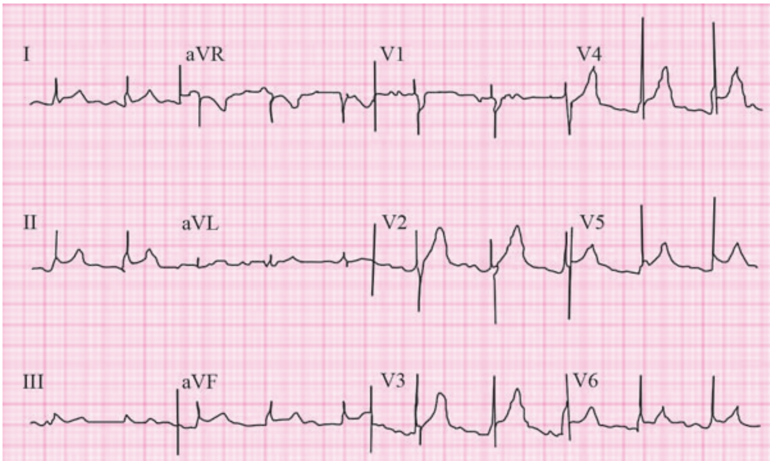


Fig. 1: *Pericarditis*: Electrocardiogram in acute pericarditis showing diffuse upstaging ST-Segment elevations seen best here in leads. II, III, aVF, and V5 to V6. There is also subtle PR-Segment deviation [positive in aVR, negative in most other leads). ST-Segment elevation is due to a ventricular current of injury associated with epicardial inflammation similarly, the PR-Segment changes are due to an atrial current of injury which, is pericarditis, typically displaces the PR-Segment upright in lead aVR and downward in most other leads.
Courtesy of Ary Goldberger, MD.

Fig. 2: Chest X-Ray showing Pericardial Effusion

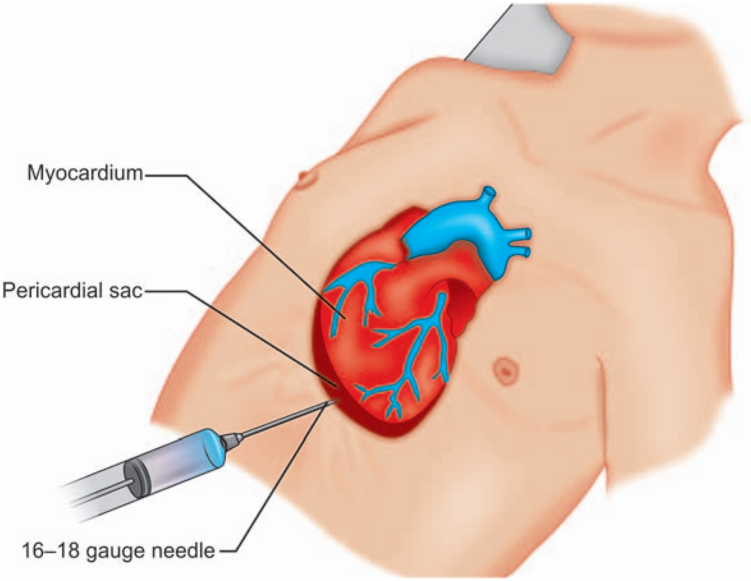


Fig. 3: Pericardiocentesis
When the needle tip enters the Pericardial sac,
withdraw nonclotted blood as much as possible under ECG guidance

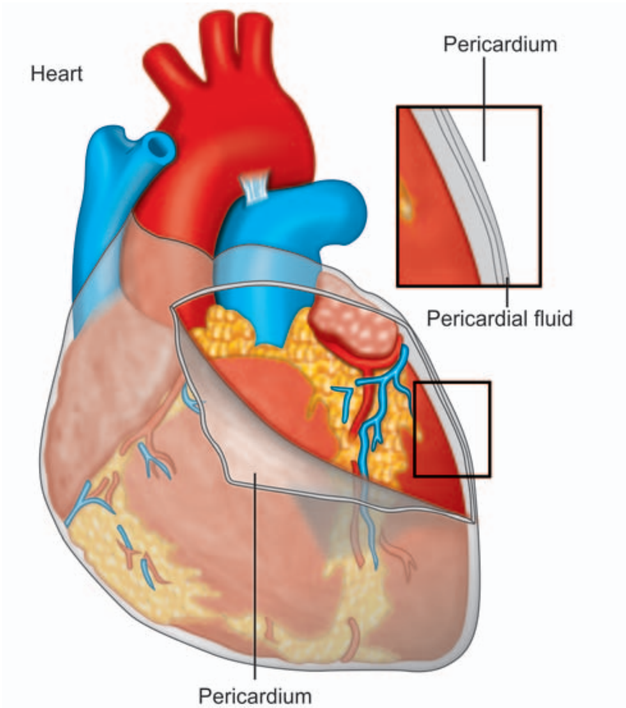


Fig. 4: Pericardium
Source: Available online from http://www.medic2010.webs.com/pericardium_580x.jpg

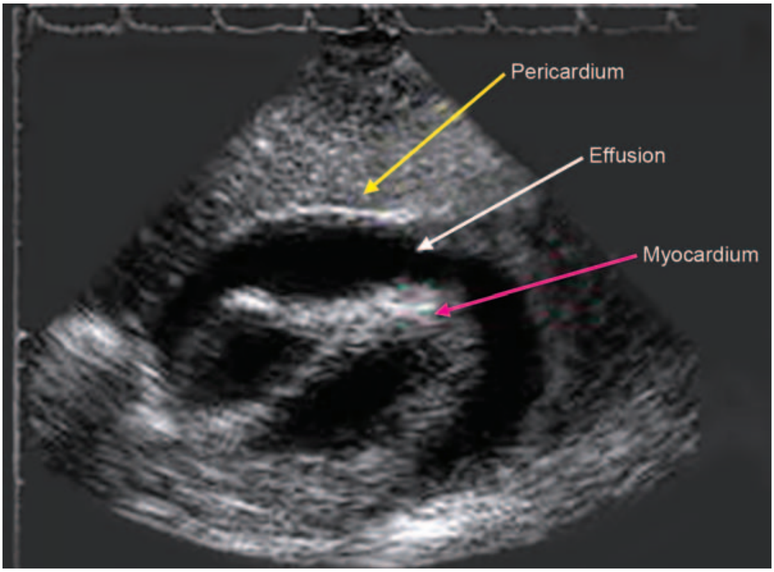


Fig. 5: Echo Pericardial Effusion

- Diagnosis of different types of pericarditis
  - Constrictive pericarditis
  - Restrictive pericarditis.

Differential Diagnoses

- *Cardiac*: Congestive heart failure
- *Infection*: Tuberculosis (TB), pneumonia
- Neoplasm

TREATMENT

- Depends on the underlying cause and may include: NSAIDs, steroids, antibiotics, etc.
- In servere situations, pericardiocentesis or surgery may be needed.

Case Study 23: Cardiac Arrest

"Saving a life is like saving a whole nation." — Al Quran

"In Cardiac Arrest your priority is CAB, not ABC; compression first please"

CASE HISTORY

A 64-year-old female was traveling from Minnesota to Chicago in a blizzard weather in December. During the boarding process, it appeared that the passenger was having seizures and was approached by the pilot. The patient was not breathing and a call for assistance was made. A fellow passenger, who is an emergency room (ER) (MD), offered to assist the pilot. Patient was pulseless and not breathing, one-person's cardiopulmonary resuscitation (CPR) was initiated until paramedics arrived on the scene with *automated external defibrillator* (AED). Weak and thready pulse palpated after 30 minutes of resuscitation. Patient was transported to the nearest hospital for evaluation. After a successful resuscitation, the pilot moved the author to the first class cabin and offered his own dinner. Later, the pilot wrote a letter to the medical director of the author's hospital praising his heroism.

TREATMENT (FIG. 1)

It involves the role of the following.

Doctor: Intubating and running the code.

Nurse: monitors vital signs and IV access, reads crash cart, places transcutaneous pacer pads (see Fig. 3) on patient, administers drugs (epi/atropine boluses), calls for electrocardiogram (ECG) and asks the charting nurse to time drugs.

Nurse Assistant: Calls the patient's family, takes history, calls the patient's private doctor and throws noncontributory people out of the room, "ALL CLEAR!" (If you're not against the wall or out of the room you might get shocked).

Respiratory equipments: Bagging with 100% oxygen, arterial blood gases (ABG) and assisting in intubation.

Laboratory technician: Drawing blood, complete blood count (CBC), calcium, magnesium, glucose and electrolytes.

Initial Survey as per the Latest American Heart Association Protocol (2010) (Fig. 2).

- Assess responsiveness (speak loudly, gently shake patient if no trauma—"Annie, Annie, are you OK?").

Fig. 1: A cardiac arrest patient's treatment

- Call for help or crash cart if unresponsive.
- Compression, airway, breathing and defibrillation (CABD)
  - Compression
    - Compressions of at least 100 per minute at a depth of 5 cm.
    - Rotate the person doing the compressions every 2 minutes.
    - See Figure 4 for 2010 AHA revised guidelines for CPR
  - Airway
    - Head-tilt chin lift (if provider suspects trauma: jaw thrust)
  - Breath—Ventilations
    - Give ventilations at a ratio of 2 ventilations every 30 compressions for one health care provider.
    - A ratio of 15:2 (compressions to ventilations) if two health care providers are present.
    - If an advanced airway is in place:
      - Give ventilations every 6–8 seconds
      - Asynchronous with chest compressions
      - About 1 second per breath
      - Visible chest rise.
  - Defibrillation
    - Attach and use AED as soon as available. Minimize chest compressions before and after shock; resume chest compressions immediately after shock.
    - Shock energy
      - Biphasic
        - Initial dose of 120–200 J (if unknown use maximum)

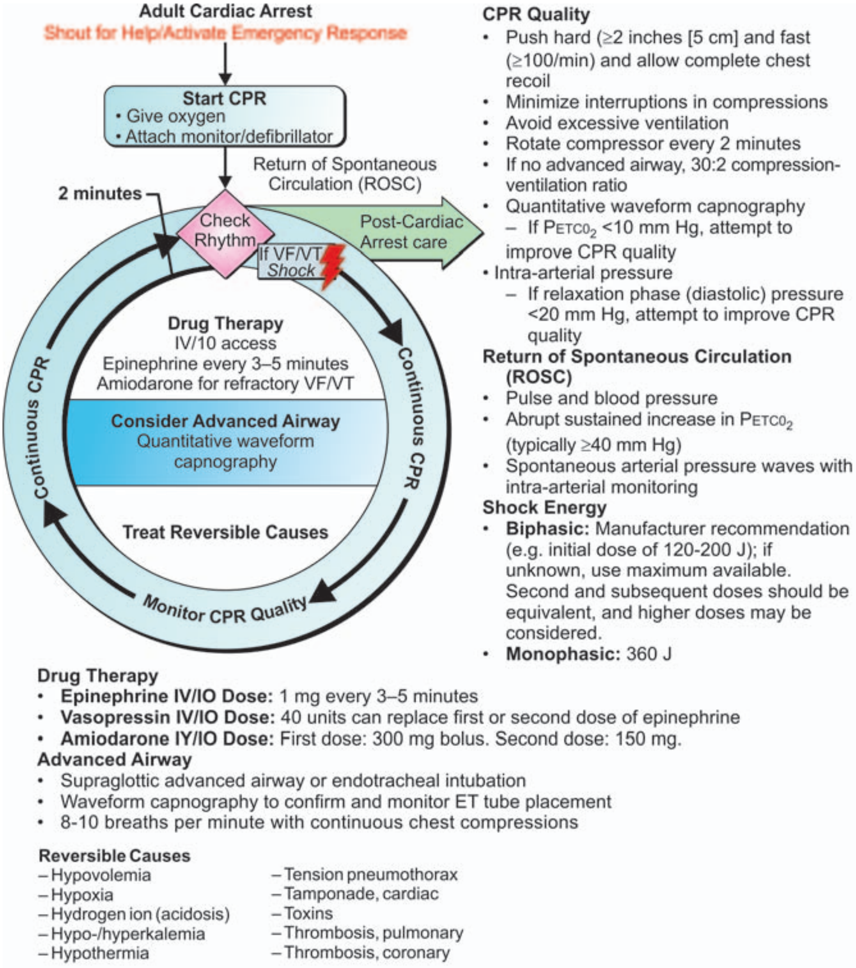


Fig. 2: Flow chart of an adult's cardiac arrest

For the most recent guidelines please contact the American Heart Association at: 1-800-242-872.

- Second and subsequent doses same as first.
- Higher doses may be considered after second dose

– Monophasic
  - One dose of 360 J

◦ Repeat the cycle.

Lastly, in developing countries there is a misconception about the use of intraosseous lines in cases where no peripheral access is available. This procedure is confined to be used only in the pediatric population, but it is wide and clear that this is the method of choice for adults as well. Using a tibial intraosseous line is found to be the most preferable life-saving intervention in the out-of-hospital cardiac arrest setting. Research done

Table 1: Factor associated with improved outcomes in cardiac arrest
• Presenting rhythm of VT/VF
• Early/bystander CPR
• Early defibrillation
• CPR prior to defibrillation in the circulatory phase of cardic arrest
• Minimal interruptions to chest compressions
• In-hospital and out-of-hospital use of AEDs
• Amiodarone use in shock-resistant VT/VF
• Therapeutic hypothermia in comatose cardiac arrest victims

Source: Avialable online from http://www.ebmedicine.net

Fig. 3: Acute cardkac arrest
Source: Available online from http://aedlifesaver.files.wordpress.com/2009/07/defibtech-defibr illation-electrode-pads.jpeg?w = 600

by Reades, Studnek, Vandeventer and Garrett (2011) states that the tibial intraosseous access had the highest first-attempt success for vascular access.[1] It also had the most rapid time to vascular access during out-of-hospital cardiac arrest when tested against peripheral intravenous and humeral intraosseous access. Table 1 shows factors associated with better outcomes in cardiac arrest.

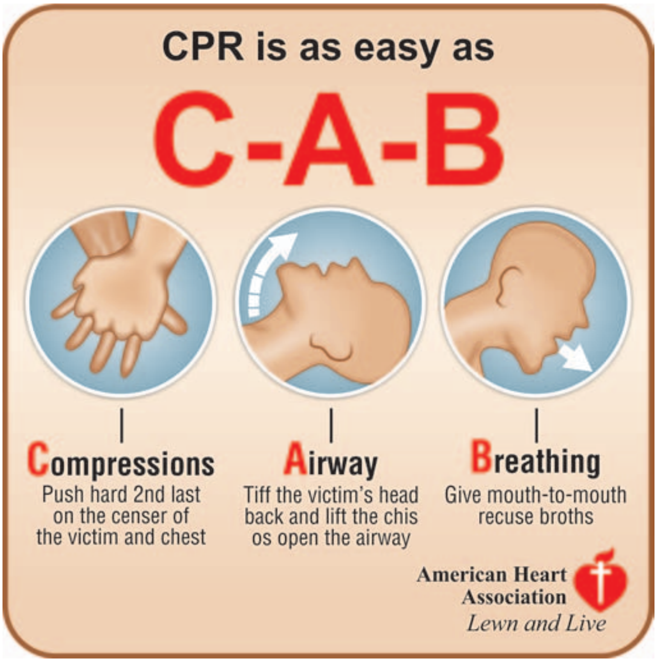


Fig. 4: A new order for CPR, spelled C-A-B[2]

REFERENCES

1. Reades R, Studnek J, Vandeventer S, et al. Intraosseous versus intravenous vascular access during out-of-hospital cardiac arrest: a randomized controlled trial. Ann Emerg Med. 2011;58(6):509-16.
2. CAB, American Heart Association http://www.heart.org/HEARTORG/

Case Study 24: Cardiac Arrest Outside Hospital: Use of Intraosseous Line

CASE HISTORY

A 55-year-old man and his wife are on vacation in Seattle. While returning home, they stop for a slice of pizza at the airport when suddenly the man passed out and is unresponsive. His wife calls for help, and shortly after, paramedics arrived. Upon arrival, the paramedics determined that the man is in full cardiac arrest so one of the paramedics started cardiopulmonary resuscitation (CPR) while the other one started an intraosseous line (IOL). The patient is successfully resuscitated and brought to the emergency room for further treatment. Upon arrival to the emergency department (ED), the man's wife states that the patient was diagnosed with coronary artery disease 4 years ago by his physician.

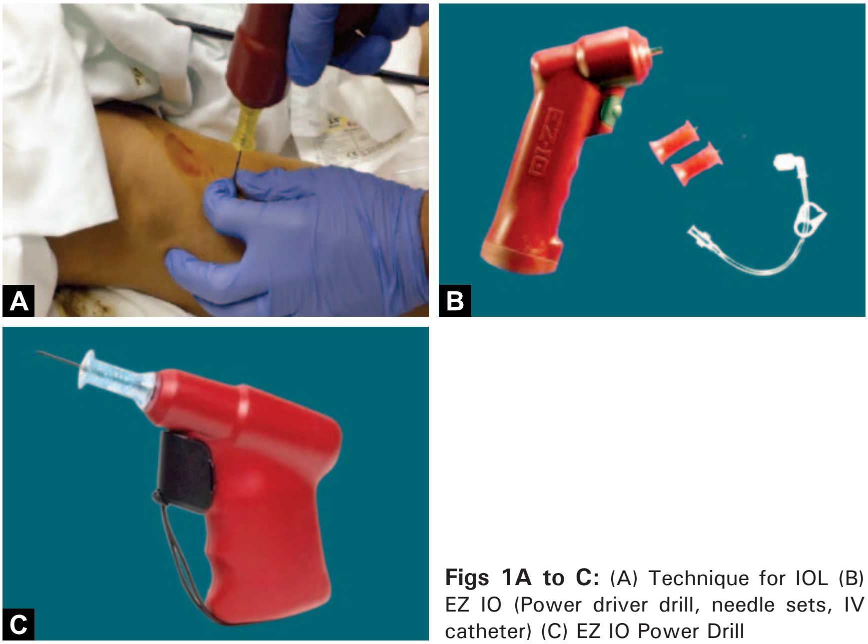
DISCUSSION

Within the past 5 years, emergency medical training companies have changed protocols to include IOLs as part of treatment for cardiac arrest with adults as well as children. When setting up an IOL on a patient, there are three preferred sites: (1) sternal, (2) tibial and (3) humeral. The sternal site makes it difficult to perform CPR at the same time and is hardly used. Until recently, there is little evidence either way as to whether the humeral or tibial site is better. Current research has shown that IOL access to the tibia has a 40% greater initial success rate than humeral IOL or peripheral intravenous sites.[1] Also, the tibial site has a lower time to needle placement and provides less needle dislodgments. Although the peripheral intravenous site is the most common site used in healthcare, it is slower to place than IOLs and still has the same issues with dislodging. Though slower and less effective, intravenous access is still by far the cheapest access method used.

INTRAOSSEOUS LINE PLACEMENT

Treatment

When treating for cardiac arrest, time is of the essence because the lack of blood circulation means that no oxygen is perfusing to the cardiac tissue. As little as 2 minutes of hypoxic conditions can cause tissue death. Immediately when paramedics arrive on the scene and suspect cardiac



Figs 1A to C: (A) Technique for IOL (B) EZ IO (Power driver drill, needle sets, IV catheter) (C) EZ IO Power Drill

arrest, one paramedic should start doing CPR while the other paramedic inserts an IOL in the tibia (Figs 1A to C). There are three important rules to remember when inserting a tibial IOL. The first is that the trapdoor effect is not seen when using the drill, the second is that the medical professional should estimate the depth needed prior to drilling. Lastly, the expert should stop sooner than they think, as you can always go back and drill deeper, if necessary.[1] Defibrillation is the next step in the treatment of this patient. Paramedics should focus on the airway, breathing and circulation (ABC) till they get to the hospital where the ED physician will order drug therapy, and more cardioversion or surgery, if necessary.

REFERENCE

1. Reades R, Studnek JR, Vandeventer S, et al. Intraosseous versus intravenous vascular access during out-of-hospital cardiac arrest: a randomized controlled trial. Ann Emerg Mee, 2011;58(6) 509516.

Case Study 25: Congestive Heart Failure

CASE HISTORY

A 32-year-old certified nursing assistant (CNA) with a history of depression had stopped taking her medication. Recent social history included divorce and a family of 5 children to care for. She was presented to the emergency room (ER) with complaints of trouble in breathing and was brought to the examination room immediately. When transferred to the bed, the patient had completely stopped breathing and was intubated. Frothy and pink secretions were assessed with the endotracheal (ET) tube. This patient was presenting with congestive heart failure (CHF) exacerbation, a portable chest X-ray was ordered and fulminant pulmonary edema was evident. An intravenous (IV) furosemide 80 mg was started and also an IV antibiotic was administered, and foley catheter was inserted with 2L of urine draining. Repeated chest X-ray showed improvement after 4 hours and patient was admitted to intensive care unit (ICU).

TREATMENT APPROACH

The primary goal of treatment in acute cases of CHF is to provide symptomatic relief. Initial management options include a combination of oxygen, morphine, diuretics, ultrafiltration, vasodilators and inotropes. Morphine results in mild vasodilatation and slows the heart rate. Morphine can be particularly useful if the patient is restless and significantly dyspneic.[2] All patients should be admitted to the hospital. If the patient responds adequately to initial treatment, telemetry monitoring is acceptable. Those who are hypotensive or fail to respond to initial therapy require admission to the ICU and may need invasive monitoring if tissue perfusion is compromised.[1] If cardiogenic shock is present, invasive evaluation is required.

Maintenance of Oxygen Saturation

Oxygen therapy should be given to all patients to maintain oxygen saturation between 95% and 98% and to maximize tissue oxygenation. Ventilation with noninvasive positive pressure ventilation (NIPPV) or continuous positive airway pressure (CPAP) may be required if oxygen saturation cannot be maintained by oxygenation alone, and is associated with a decreased requirement for intubation and mechanical ventilation. Mechanical ventilation is only used when other treatments, including noninvasive ventilation methods, fail.

Hemodynamically Stable

Diuretics and Vasodilators

- Loop diuretics are the mainstay of treatment and are effective in relieving symptoms. Non-loop diuretics, such as spironolactone and metolazone, may be added if there is an inadequate response to loop diuretics alone. Intravenous diuretics are indicated in patients with a systolic blood pressure (BP) higher than 85 mm Hg.
- Vasodilators (glyceryl trinitrate, nitroprusside and neseritide) are indicated in patients with pulmonary congestion/edema and a systolic BP higher than 90 mm Hg. Glyceryl trinitrate is the first-line agent with nesiritide considered second-line.
- Although there are no large-scale studies comparing diuretics alone with glyceryl trinitrate in patients with acute CHF, some have suggested that nitrates alone may be a better alternative in patients with acute CHF. In clinical practice, both these agents are used in combination. [Level B Evidence]

In patients who do not respond to initial therapy, extracorporeal ultrafiltration is used to reduce volume overload. [Level A Evidence]

Hemodynamically Unstable

Patients with hypotension or hypoperfusion (i.e. cold and dry, cold and wet profiles) should be commenced on inotropic support as this may improve hemodynamics. [B Evidence] However, positive inotropes should be used with caution because there is evidence that they result in increased mortality and can cause arrhythmias and worsening of coronary ischemia. [B Evidence] The occurrence of sustained arrhythmias should lead to their discontinuation. Concomitant use of amiodarone may be advisable, although there are no large-scale data on the use of antiarrhythmics in this setting. If the patient has symptomatic coronary ischemia, inotropes should be discontinued.

Patients with a systolic BP below 90 mm Hg or a drop of mean arterial pressure of more than 30 mm Hg with a pulse rate above 60 beats/minute and/or low urine output (< 0.5 mL/kg/hour) are defined as being in cardiogenic shock. Insertion of an intra-aortic balloon pump is indicated in patients with persistent cardiogenic shock, despite inotropic therapy. However, patients with significant aortic regurgitation or aortic dissection are not candidates.

Choice of inotrope depends on clinical findings.[1] Dobutamine or milrinone are recommended for patients with a systolic BP of 85–100 mm Hg and no clear clinical evidence of shock, such as cold extremities and low urine output. Levosimendan, a calcium sensitizer, may be used as an alternative to dobutamine or milrinone. It may not be available in

some countries. Dopamine is recommended for patients with systolic BP below 85 mm Hg with clinical evidence of shock.[1] Norepinephrine (noradrenaline) is recommended for patients with systolic BP below 85 mm Hg and persistent signs of shock.

Specific Treatment of Underlying Cause

Coronary Artery Disease

- Intravenous glyceryl trinitrate (nitroglycerin) is first-line treatment.
- The common adverse effect of glyceryl trinitrate is headache and hypotension. The dose of nitrates should be reduced if systolic BP decreases below 90–100 mm Hg and discontinued permanently if BP drops further. From a practical point of view, a reduction of 10 mm Hg in mean arterial BP should be achieved.
- In cases of significant coronary artery disease (CAD) causing acute CHF, percutaneous revascularization or coronary artery bypass should be carried out. Aspirin is given to all patients with coronary ischemia and those undergoing revascularization.
- In the case of cardiogenic shock with acute myocardial infarction (MI), revascularization is recommended. Thrombolysis in this setting is not effective.

Hypertensive Emergency

- Use of IV beta-blockers and glyceryl trinitrate is recommended.
- If additional medicines are needed, nitroprusside is recommended in addition to other choices.

Valvular Heart Disease

- In cases of severe aortic stenosis with heart failure, nitroprusside can be used provided the patient is not hypotensive.
- The definitive treatment for aortic stenosis is transcatheter aortic-valve implantation (TAVI) if the patient is not a suitable candidate for conventional surgery. Mitral stenosis, if severe, needs a valve replacement, but in resistant heart failure a percutaneous valvotomy may be used as temporary measure until definitive valve replacement is carried out. In mitral stenosis, percutaneous valvuloplasty may be done if no thrombus is present on transesophageal echocardiogram (TOE).
- Similarly in heart failure associated with mitral regurgitation or aortic regurgitation, a vasodilating drug such as nitroprusside should be used. A decrease in the peripheral arterial resistance results in an increase in the cardiac output and a decrease in regurgitant volume which in turn is associated with a reduction in left ventricular end-diastolic volume and an augmentation of the ejection fraction.

Acute Right Heart Failure

- Treatment is focused on the underlying pathology; e.g. pulmonary embolism (anticoagulation, thrombolytics, catheterization or surgically directed thrombectomy), right ventricular infarction [percutaneous coronary intervention (PCI) or thrombolytics], and chronic thromboembolic pulmonary hypertension (thromboendarterectomy).

Acute Myocarditis

- Giant cell myocarditis is treated with single or combination of immunosuppressant therapy including corticosteroids, azathioprine, cyclosporine and muromonab-CD3 (OKT3).
- Treatment of other forms of myocarditis is limited to supportive care.

Resistance to Maximal Medical Therapy

In cases of resistance to maximal medical therapy, a left ventricular assist device (LVAD) should be inserted. In some cases of nonischemic cardiomyopathy, sustained reversal of severe heart failure is seen with implantation of an LVAD. The use of LVADs has evolved significantly over the past 25 years and various types of LVAD now exist. Extracorporeal devices, the most common of which are the extracorporeal membrane oxygenators (ECMOs), require full heparinization and are typically used for days or weeks as a bridge for patients who are expected to recover within days. Percutaneous short-term devices (e.g. Tandem Heart) are inserted through the femoral artery and advanced into the left ventricle. Longer-term assist devices are divided into first generation (e.g. Heart Mate I), second generation (e.g. Heart Mate II), and third generation (e.g. HVAD™ and Dura Heart®) devices. The third generation pumps are thought to last as long as 5–10 years and are currently being evaluated in several phase 1 studies.[1]

Ongoing Therapy

Once the patient is stabilized, definitive medical therapy for heart failure should be commenced. Usually an angiotensin-converting enzyme (ACE) inhibitor [A Evidence] (or an angiotensin-II receptor antagonist if ACE inhibitors are not tolerated) [B Evidence] is started first, followed by the addition of beta-blockers. [A Evidence] The dose of ACE inhibitors and beta-blockers should be increased to the maximum tolerated dose depending upon BP and heart rate. Patients who have persistent signs of fluid overload will need ongoing diuretics. Patients with ongoing symptoms, despite this therapy, should be treated as having chronic CHF. Probability of CHF within 4 years for women aged 45–94 years with coronary disease, hypertension, or valvular disease.

* Model including vital capacity and chest X-ray results

Points	0	1	2	3	4
Age (Years)	45–49	50–59	60–69	70–79	80–89
Forced vital capacity, (cL)	> 299	250–299	200–249	150–199	< 150
Systolic blood pressure, (mm Hg)	< 130	130–189	> 189		
Heart rate (bpm)	< 55	55–79	80–99	> 99	
LVG on ECG	No				
Coronary Heart Disease	No				
Cardimegaly	No		Yes		
Valve Disease	No				
Diabetes (no valve disease)	No				
Diabetes (valve disease)	No			Yes	

Points	5	6	7	8	9
Age, Years	90–94				
Forced vital capacity, (cL)					
Systolic blood pressure, (mm Hg)					
Heart rate bpm					
LVH on ECG	Yes				
Coronary Heart Disease					
Cardiomegaly				Yes	
Valve Disease			Yes		
Diabetes (no valve disease)			Yes		
Diabetes (valve disease)					

Points	4-Year Probability (%)	Points	4-Year Probability (%)
5	< 1	26	18
10	1	27	21
12	1	28	24
14	2	29	28
16	3	30	32
18	4	31	37
20	6	32	42
22	9	33	46
24	13	34	51
25	15	35	56

Points	0	1	2	3	4
Age, Years	45–49	50–54	55–59	60–64	65–69
BMI kg/m^2	< 21	21–25	26–29	> 29	< 150
Systolic blood pressure, (mm Hg)	< 140	140–209	> 209		
Heart rate (bpm)	< 60	60–79	80–104	> 104	
LVH on ECG	No				
Coronary Heart Disease	No				
Valve Disease	No				
Diabetes (no valve disease)	No				
Diabetes (valve disease)	No		Yes		

Points	5	6	7	8	9
Age, Years	70–74	75–79	80–84	85–89	90–94
BMI, (kg/m^2)					
Systolic blood pressure, (mm Hg)					
Heart rate (bpm)					
LVH on ECG	Yes				
Coronary Heart Disease		Yes			
Valve Disease		Yes			
Diabetes (no valve disease)		Yes			
Diabetes (valve disease)					

Points	4-Year Probability (%)	Points	4-Year Probability (%)
5	< 1	19	14
10	2	20	17
11	2	21	21
12	3	22	25
13	3	23	30
14	4	24	36
15	5	25	42
16	7	26	48
17	9	27	54
18	11	28	60

LVH indicates lelt ventricular hypertrophy; ECG, Electrocardiogram. Probiability of congestive heart failure within 4 years for women agreed 45 to 94 years with coronary disease, hypertension, or Valcular disease Model without vital capacity and chest X-ray results.

REFERENCES

1. bestpractice.bmj.com
2. books.mcgraw-hill.com

Case Study 26: Brugada Syndrome

CASE HISTORY

A 35-year-old male who has no history of cardiac risk factors is brought to the emergency room unresponsive. When hooked to the cardiac monitor, ventricular fibrillation (V-fib) is present. After 3–4 shocks at 360 J, his rhythm is restored.

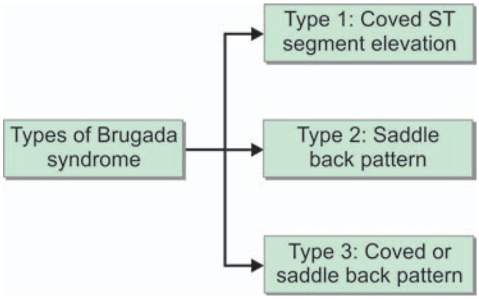
DISCUSSION

Brugada syndrome is a life-threatening heart rhythm disorder characterized by sudden death. It is a genetic disease causing abnormal heart rhythm due to the heart receiving excess electrical current. It was first identified by the Brugada brothers in a young child with abnormal electrocardiogram (ECG). It is a major contributor to causes of death in children. In fact, it may explain some cases of sudden infant death syndrome (SIDS). This syndrome may present at any age from infancy to adulthood. It is inherited in an autosomal dominant manner. Patients inherit an abnormal *SCN5A* gene which alters Na^+ channel production and function. Because of this mutation, Na^+ channel function is altered which leads to decreased flow of Na^+ ions and therefore an irregular rhythm. Early repolarization occurs. This early repolarization may lead to malignant ventricular tachycardia which leads to ventricular fibrillation and possible sudden cardiac death (SCD). Symptoms may include syncope, shortness of breath, and SCD. Diagnosis is made by ECG and echocardiography (ECHO). ECG may show elevated ST segments and inverted T waves. The current treatment is *implantable cardiac defibrillator* (ICD) placement to prevent SCD.

There are three types of Brugada syndrome. Type I has at least 2 mm ST-segment elevation either spontaneously or induced with ajmaline/flecainide (a diagnostic test). Type II has a saddle back pattern with at least 2 mm J-point elevation and ST elevation. This type also shows a positive or biphasic T-wave. Type III resembles either the shape of Type I or Type II, except the elevation of the J-point is less than 2 mm, also with less than 1 mm ST elevation. Types II and III can be seen in healthy subjects (Flow chart 1 and Fig. 1).

Brugada syndrome is the most common cause of death in young men with no known underlying risk factors in Cambodia and Laos.

Flow chart 1: Types of Brugada syndrome



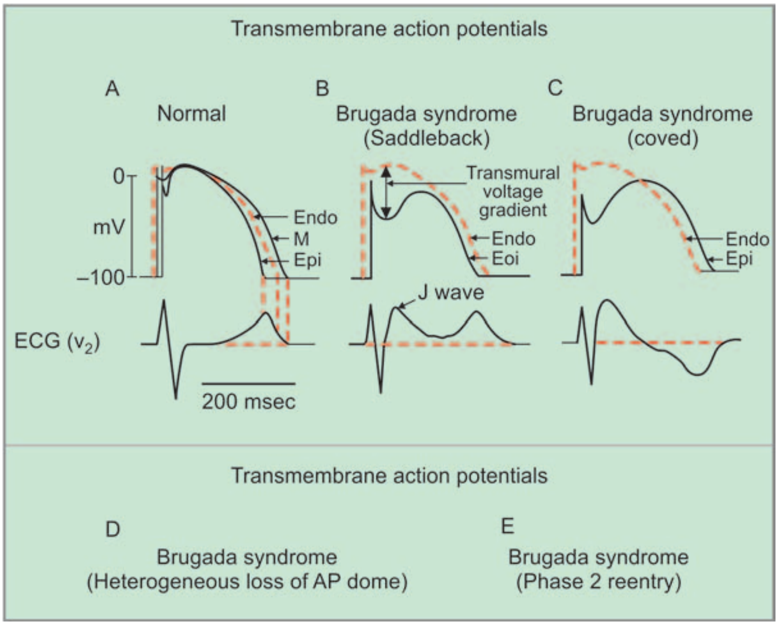


Fig. 1: Transmembrane action potentials

ADDITIONAL READING

1. For more information visit http://www.youtube.com/watch?v=eTDnEUEYlis&feature=related

Case Study 27: Hypertensive Urgency versus Emergency

"... I will keep them from harm and injustice."

—Hippocratic Oath

CASE 1: HYPERTENSIVE URGENCY

A 60-year-old diabetic male presents to the emergency room with a chief complaint of headache. The patient was alarmed, when taking his blood pressure at home, that the reading was 200/110 mm Hg. His past medical history is significant for diabetes, hypertension and chronic kidney disease (CKD) stage 3, with a *glomerular filtration rate* (GFR) between 30 and 59 mL/min for the past 6 months. He was diagnosed to have nephrosclerosis. The patient has been hypertensive for the past 20 years, but recently he has been having difficulty controlling his blood pressure. Presently, he is on three different medications: lisinopril 20 mg, amlodipine 10 mg, *hydrochlorothiazide* (HCTZ) 25 mg. His lab tests show proteinuria and a spot protein-urine sample shows urine protein excretion is less than 500 mg/day. Physical examination shows body mass index (BMI) of 32, but does not reveal any evidence of abdominal bruit. Also, there is no mental status change and no significant neurological deficits. Funduscopic examination of the eye shows no diabetic retinopathy and no hypertensive retinopathy. Ultrasound examination shows bilateral renal disease without obstruction. Kidney size is 9.1 cm right and 9.6 cm left. This case represents hypertensive urgency (Flow chart 1).

PATIENT'S MANAGEMENT

Cautious reduction of blood pressure within 24–48 hours with oral medications can be achieved. You can do more harm to a patient with hypertensive urgency by aggressively reducing the blood pressure. You may complete angina to full-blown acute myocardial infarction (AMI), or you may complete a partial stroke/stroke in evolution to complete stroke.

CASE 2: HYPERTENSIVE EMERGENCY

A 77-year-old male is brought to the emergency department by the paramedics after a sudden onset of right-sided weakness associated with difficulty of speaking. His wife called the ambulance when she observed him returning from the washroom with right-sided weakness and he was slurring his speech. Past medical history is significant for poorly-controlled hypertension and diabetes secondary to poor

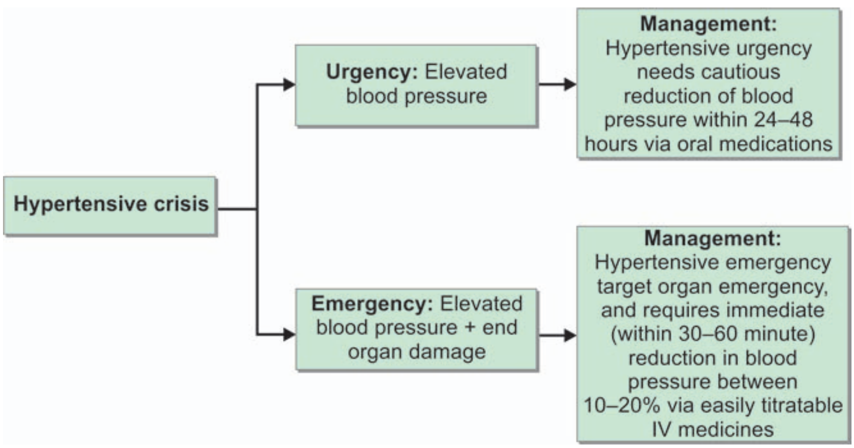
compliance, and there is no history of atrial fibrillation (Flow chart 2). He had a hemoglobin A1c drawn last week which showed 8%. Diagnostic tests; CT head (noncontrast) shows multiple old lacunar infarcts and parietal ischemic stroke. On arrival to the emergency department, his vitals are blood pressure of 200/110 mm Hg, pulse rate 100 beats/minute, temperature 36.9°C, and complete blood count (CBC) and chemistry profile within normal limits (WNL).

PATIENT'S MANAGEMENT

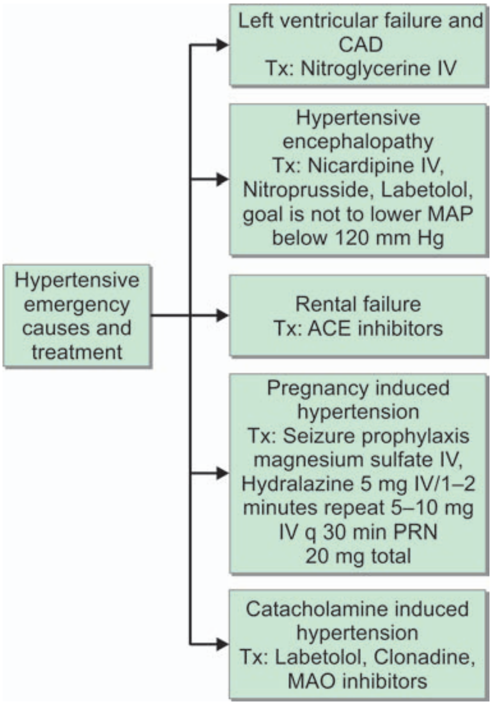
This is a true hypertensive emergency where elevated blood pressure has caused end-organ damage/dysfunction (Figs 1A to D). In this scenario, the ischemic injury needs to be treated very cautiously and slowly. Since this is a target organ injury to the brain, hence, cerebral autoregulatory ability is disrupted. In this situation, there are two causes of injury to the patient; ischemia and decrease in cerebral blood flow. A variety of studies have shown a relationship between systolic BP reductions and stroke outcomes. The higher the blood pressure the better the outcome. Another study has shown that a poor outcome was independently associated with the degree of systolic blood pressure reduction. As a result of varied data from different studies, a scientific statement from the stroke council of the American Stroke Association has recommended that blood pressure, generally, not be lowered in patients suffering from acute ischemic strokes who are not otherwise candidates for thrombolysis. Mainly treating the cause of hypertension is very helpful.

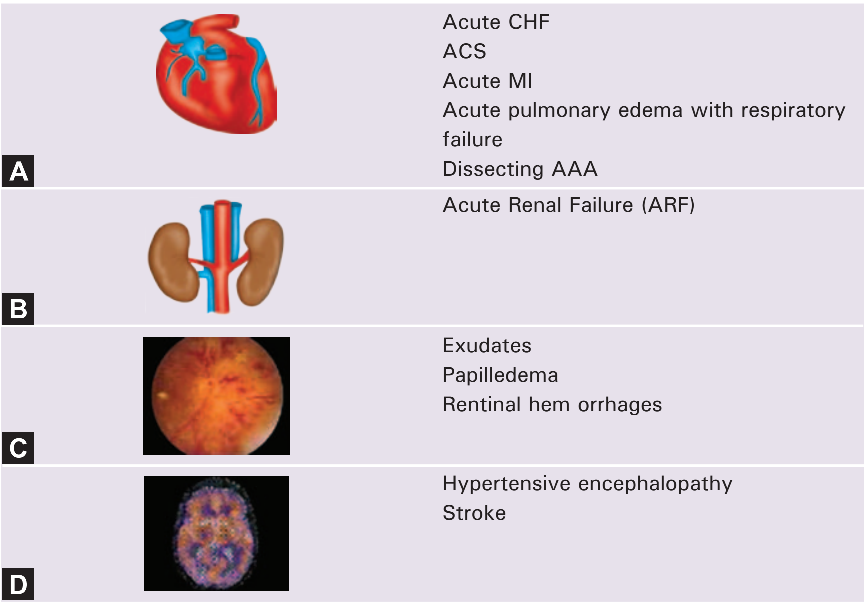
DISCUSSION

Flow chart 1: Hypertensive crisis



Flow chart 2: Causes and treatment of hypertensive emergency





Figs 1A to D: End-organ damage in hypertensive emergency

ADDITIONAL READING

1. http://www.researchgate.net/

Case Study 28: Left Ventricular Assist Device

"Except for the occasional heart attack, I never felt better."

—Dick Cheney

CASE HISTORY

A 55-year-old man is on a business trip from Seattle to a rural area in Southern Illinois. He is out to dinner and starts experiencing chest pain and throws up bright red blood, so his co-workers call the ambulance. While riding in the ambulance he insists that he did not receive cardiopulmonary resuscitation (CPR) and gives a card to the paramedics about a left ventricular assist device (LVAD) and a doctor's name. While talking to the patient, the paramedics cannot find a pulse on the patient's body. By the time they get to the hospital, the patient is unconscious but his orders are transferred from the paramedics to the emergency department (ED) doctor. None of the ED doctors has heard of an LVAD so the patient's cardiologist on the card is called.

DISCUSSION

Left ventricular assist devices have been around for almost 20 years, but are not widely used since heart transplant is still the gold standard. The first generation devices used a pulsating flow to push the blood in waves out of the ventricle into the aorta. Current devices (second generation) use a continuous flow to keep blood moving, but can also cause more alarm to an ED doctor as a patient may present without a pulse but still able to talk! To qualify for an assistive device [right ventricular assistive device (RVAD) and both ventricular assistive device (BiVAD) also exist], the patient must have Stage IV New York classification heart failure and an ejection fraction of 25% or less.[1] Most of the time these devices are used to keep a person alive until a transplantation can be found. In some cases, they are used as a destination treatment for heart disease, because most patients do not fit the specific criteria for transplantation.

Patients with heart failure are often limited in what they can do and often not able to accomplish many activities of daily living. After having a LVAD inserted, most patients go from Stage IV heart failure to Stage I or II and regain much functioning (with the exception of swimming). The longest lasting second generation device to date has been 6 years, but there is no reason they cannot last longer. In addition, if problems with the pump happen, the device should be able to be replaced as many times as necessary limited only by patient healing (Fig. 1).

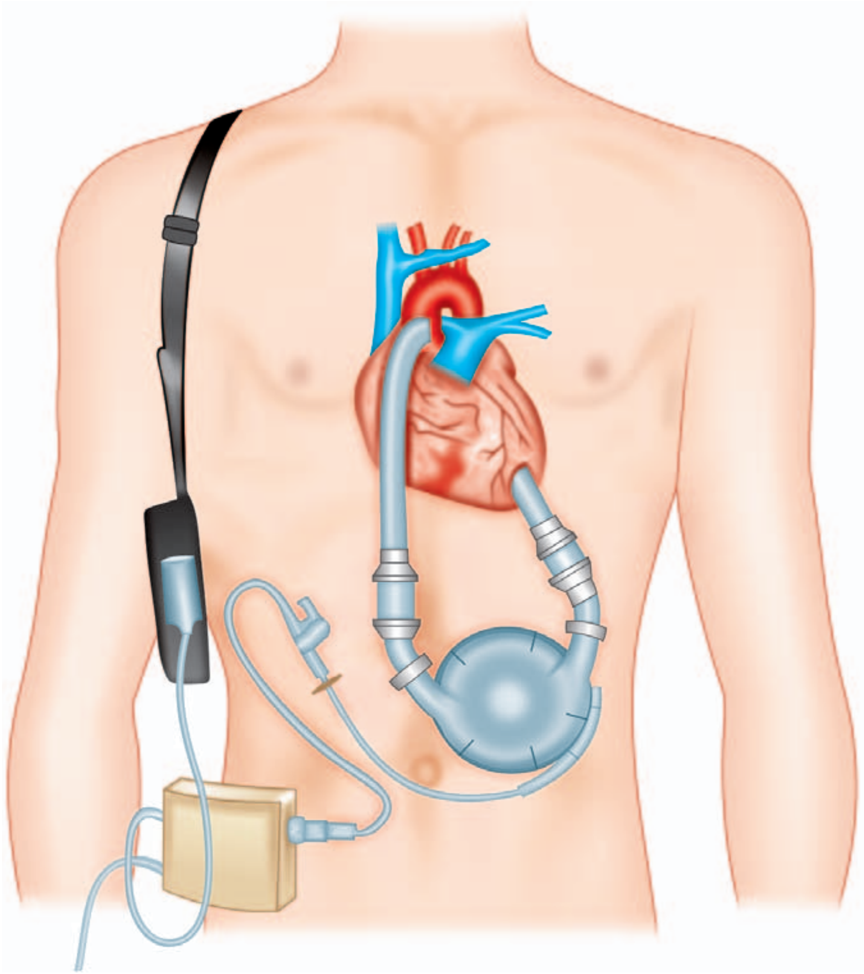


Fig. 1: A left ventricular assist device (LVAD) pumping blood from the left ventricle to the aorta, connected to an externally worn control unit and battery pack

TREATMENT

Until recently we have only seen people coming for dobutamine IV weekly for treatment of congestive heart failure (CHF), but since the advance of modern medicine LVADs have replaced such treatments (Table 1). There are four very distinct reasons why medicine is moving toward LVAD implantations which include; limited need for medication, less follow-up, fewer invasive procedures, and they still have the option for transplant. Former Vice-President of America, Dick Cheney, had LVAD implanted.

Table 1: Complications from left ventricular assistive device and its treatment

Complications from left ventricular assistive device	*Treatment*
Infection/cellulitis	Hospitalize and give IV antibiotics
Bleeding	Look and treat for coagulopathy
Pulselessness/no heart sounds	Listen to precordium for humming sound of the device. Examine power supply and battery, change if necessary and call the control center. Place the patient on cardiac monitor and obtain Doppler blood pressure.
Ventricular dysrhythmias	Defibrillate
Hypoxia/acidosis	Intubation and administer $NaHCO_3$ IV
Right ventricular failure from thrombosis in left ventricular assist device or pulmonary embolism	Anticoagulation (heparin) therapy used for non-bleeding patients.

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CHAPTER

3

Abdomen

Case Study 29: Upper Gastrointestinal Bleed

CASE HISTORY

A 45-year-old investment banker presents with an episode of black vomit. He has been experiencing stomach pains for weeks, often after he finishes a meal. He wakes up at night with burning chest pain and describes a foul taste in the morning. The patient also complains of bloating and fullness. His job has been stressful, as the economy has been taking massive downturns. He describes himself as an easily stressed person. The patient has had tension headaches for many years that he usually responded by using a few capsules of Advil. The patient also admits that he has been feeling weak and a reduced sex drive.

PHYSICAL EXAMINATION

Patient is tachycardic. Abdomen examination shows tenderness in the epigastrium.

Laboratory Tests

Low hematocrit. Hypochromic anemia.

Imaging

Endoscopy: Gastric ulcer

DIAGNOSIS

The patient is suffering from upper gastrointestinal (GI) bleeding secondary of nonsteroidal anti-inflammatory drug (NSAID) overuse.

DISCUSSION

Before the details of the case are discussed, it is helpful to define a number of terms:

- *Hematemesis*: Vomiting of blood.

- *Coffee ground emesis*: Vomiting of blood that has been broken down by stomach acid, which shows the color of oxidized heme.
- *Hematochezia*: Presence of fresh blood in stool.
- *Melena*: Presence of digested blood in stool.

This is a case of upper GI bleeding. The presence of "coffee ground" emesis, epigastric discomfort, and history of stress and NSAID use highly suggest an upper GI bleed before endoscopic confirmation of a gastric ulcer. Upper GI bleed refers to any bleeding above the ligament of Treitz of the duodenum. The form of blood in either emesis or stool can give clinicians clues as to where the location of bleeding may be, even within the scope of upper GI bleeding. In the case of "coffee ground" emesis, an upper GI bleed is most likely. The bleed is unlikely to be in the esophagus due to its oxidized nature. If it is a gastric bleed, the bleeding may be low-grade, which allows blood the time to be oxidized. In contrast, the presence of hematemesis suggests bleeding in the esophagus or heavy bleeding in the lower upper GI tract.

The forms of blood in stool are also helpful in diagnosing GI bleeds. In the case of melena, the bleeding location is likely to be upper GI due to the fact that the blood had the time to pass through the GI system to be digested. If hematochezia is present, the bleeding is likely to be in the lower GI due to its proximity to the anus. Hematochezia may also occur with severe upper GI bleeding, in which the volume of blood overcomes the digestive capacity of the system. In such cases, the patients are likely to exhibit signs of hypovolemia, anemia and even shock.

There are many causes of upper GI bleeding. Gastric ulcer secondary to NSAID use is a prevalent problem. NSAIDs inhibit cyclooxygenases, which are responsible for producing prostaglandins that protect the gastric mucosa. Another common cause of gastric ulcer is *Helicobacter pylori (H pylori)* infection. The bacterium erodes the mucosa, allowing gastric acid to further damage the underlying gastric and duodenal tissues. Ulcers may also result from heavy alcohol consumption, in which the alcohol irritates the gastric mucosal lining and causes acid damage. Other causes of upper GI bleeding include Mallory-Weiss syndrome, cancer, and esophageal/gastric varices.

The diagnosis of GI bleeding is through upper endoscopy. Endoscopy can verify the presence and site of the bleed and perform coagulation therapy (Fig. 1). Nasogastric tube may also be used to aspirate gastric content to determine if blood is present (to differentiate between lower GI bleeding). Other diagnostic tests, such as hemoglobin, hematocrit, colonoscopy, and stool occult blood are also helpful in differentiating upper GI bleed from other diagnoses.

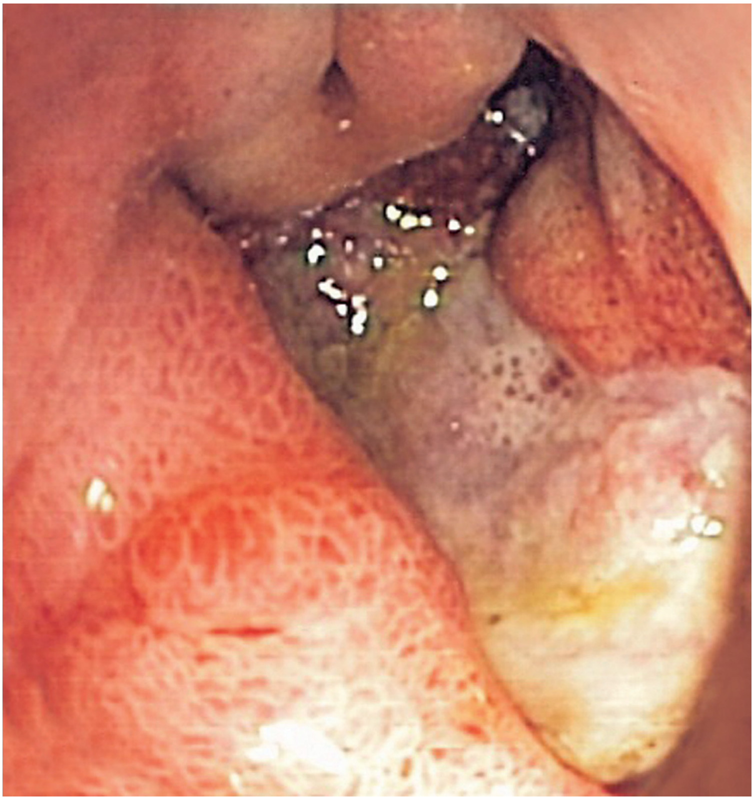

Fig. 1: Endoscopy showing deep gastric ulcer

Source: http://www.cleanandsoberlive.com/stomach-pain-after-drinking-its-probably-gastritis/

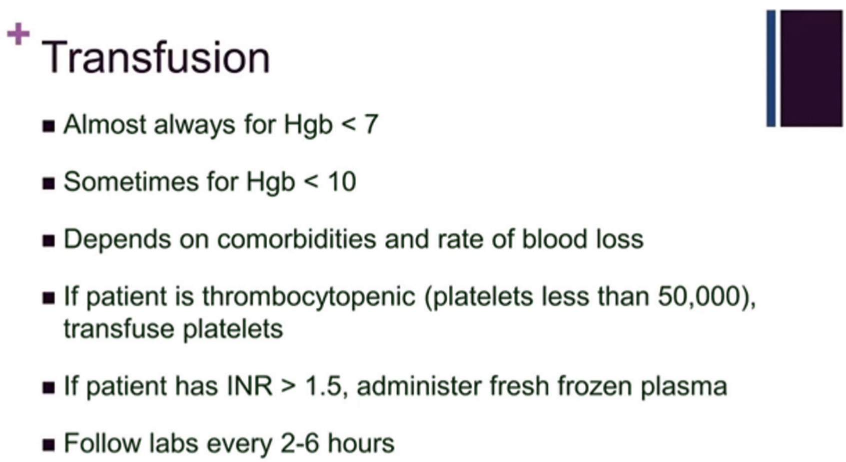

Fig. 2: Criteria for transfusion of blood

Source: Gastriuntestinal Emergencies, 2nd Edition. ISBN:978-1-405-14634-0

The first priority of treatment of an upper GI bleed is resuscitation and stabilization if the patient is hemodynamically compromised. The subsequent management depends on the severity and the underlying cause of bleeding. Active bleeding is treated with endoscopy with coagulation to stop the bleed. If the bleeding cannot be stopped with endoscopic procedures, surgery would be the next step. Pharmacological interventions may follow to treat the underlying cause of the bleed. In the case of *H pylori* infection, a proton pump inhibitor and combinations of antibiotics are necessary. Octreotide is used to treat esophageal or gastric varicies.

Depending on the comorbidities and rate of blood loss, transfusions are almost necessary if the Hgb is < 7.

1. Sometimes when Hgb < 10, patient may require transfusions
2. If patient is thrombocytopenic, they may require platelet transfusion
3. If patient has INR > 1.5, you should administer fresh frozen plasma
4. Continue to follow labs ever 2–6 hours.

For very severe hemorrhage, prophylactic antibiotics can be administered to reduce the morality.

REFERENCE

1. Gastriuntestinal Emergencies, 2nd Edition. ISBN:978-1-405-14634-0

Case Study 30: Diverticulitis

"If you believe the patient has appendicitis on the left side, you are most likely dealing with diverticulitis"

—Badar M Zaheer

CHIEF COMPLAINT

An 84-year-old postmenopausal white female with abdominal and flank pain.

CASE HISTORY

An 84-year-old lady with a history of smoking presents to the ER at 5:50 am with sudden constant left lower quadrant (LLQ) pain that radiates to the groin with an onset of 3 hours. Current pain level is 2–3/10 and maximum was 10/10. Last bowel movement was 20–25 minutes prior to arrival which was normal but thinner. She presents with a mild fever, but no chills, sweating, chest pain, nausea, and vomiting. No diarrhea or constipation were noticed. Nothing seems to exacerbate or relieve her symptoms.

PAST SURGICAL HISTORY

Right hip surgery.

DIAGNOSTIC TESTS

- CBC—Patient has 16,000 WBC count with shift to the left.
- Abdominal X-rays—Not useful.
- Barium Enema—Outdated and not useful.
- CT abdomen—Preferred choice. Highly sensitive and specific (Figs 1 and 2).
- Ultrasound—Safe, less invasive, low cost, but operator dependent and not as accurate as CT. Not a useful tool for surgical planning.

CASE DISCUSSION

Many people have small pouches in the lining of the large intestine that bulge outward through weak spots. The small pouches are called diverticula, and the condition of having diverticula is called diverticulosis. As we age, this condition becomes more prominent.[1]

In 10–25% of people with diverticulosis, the pouches become inflamed. Acute diverticulitis is the most common complication of diverticular disease, but 20% of the patients with diverticular disease may also

Fig. 1: CT abdomen with sigmoid wall thickening and adjacent fat stranding
Source: Badar M Zaheer MD

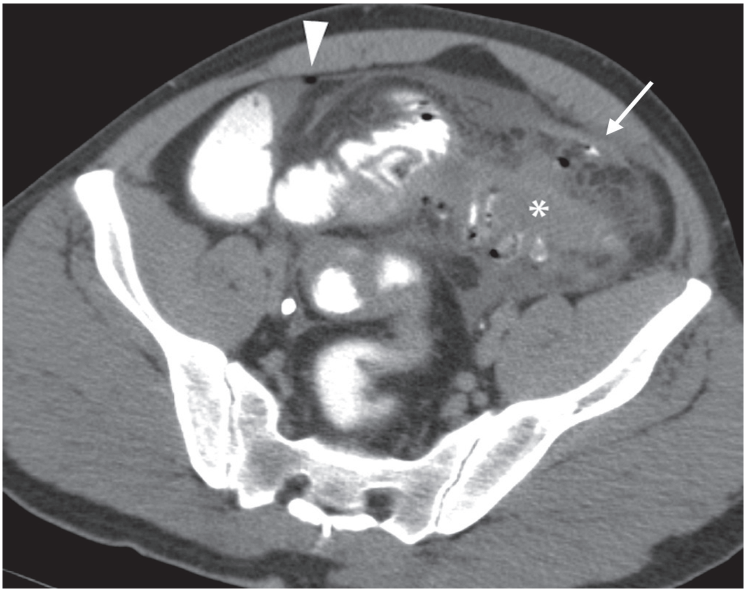

Fig. 2: Image shows severe diverticulitis with perforation. This patient presented with abdominal pain, fever, and rebound tenderness
Source: Badar M Zaheer MD

develop other complications, such as infection, abscess, fistula, obstruction, perforation, or even diverticular bleeding. Patients usually

present with visceral abdominal pain and tenderness localized to the area of maximal inflammation. Nausea, vomiting, and altered bowel habits often occur. There may also be rectal tenderness, fever, and leukocytosis.

MANAGEMENT

In mild cases where the patient has the ability to tolerate oral intake, he or she can be treated as an outpatient. Put the patient on clear liquid diet and a broad spectrum antibiotic for 7–10 days (amoxicillin-clavulanate, trimethoprim-sulfamethoxazole, or fluorquinolone + metronidazole), and discharge home for a close follow-up.

Patients suffering from a severe attack of diverticulitis may require hospitalization. The patient must be put on analgesia, bowel rest, intravenous fluid, and a broad-spectrum antibiotic for anaerobes and gram-negative rods for 7–10 days. Anaerobes are treated with metronidazole or clindamycin. Gram-negative rods are treated with aminoglycoside, monobactam, or third-generation cephalosporins.

Pericolic abscesses are drained under CT or US guidance. The patient may also be treated with conservative methods, as those stated above.[2]

Treatment focuses on clearing up the inflammation and infection, resting the colon, and preventing or minimizing complications. Gradually increasing the amount of high-fiber foods in the diet will help in recovery.

PHYSICAL EXAMINATION (07:37 AM)

General Appearance

The patient is alert, oriented, and not in acute distress.

Head, Eyes, Ears, Nose and Throat (HEENT)

There is no evidence of trauma, tumors, facial edema, goiter or thyroid nodules. Pupils are equal, round, reactive to light and accommodation (PERRLA) and sclera anicterus. ENT inspection is normal. Pharynx is normal. The neck is supple.

Cardiovascular System

Regular rate and rhythm. S1 and S2 are normal.

Respiration

Breath sounds are normal.

Table 1: Medications				
Time	*Medication*	*Dose (mg)*	*Route*	*Site*
08:07	Zofran	4	IV	LW
08:13	Morphine sulfate	2	IV	LW
08:45	Nitro patch	0.4	TOP	Left Chest
09:45	Ativan	0.3	IV	LW
10:06	Zofran	4	IV	LW
10:20	Compazine	5	IV	LW
11:25	Tylenol	650	PR	
12:05	Motrin	400	PO	

Table 2: IV Medication Infusion				
Time	*Solution/Medicine Type*	*Rate (mL/hr)*	*Stop Time*	*Amount Infused*
10:20	0.97 NS1 Med.	200		
10:25	Rocephin, 1 GM 1 Med.	200	10:50	100
10:51	Gentamicin, 125 mg 1 Med.	300	11.20	100
11:20	0.9% NS Pump	999	11:45	300
11:46	0.9% NS Pump	999	11:59	300

Abdominal

- Soft, tenderness in LLQ
- Normal bowel sounds. No abdominal bruit. No pulsatile mass. No Hepatosplenomegaly.

Skin

Color is normal. No rash. Warm and dry.

Extremities

Nontender, Normal range of motion (ROM), no pedal edema, no joint effusions, rashes, or cyanosis.

Lab

Complete blood count (CBC), comprehensive metabolic panel (CMP), amylase, lipase, blood culture and sensitivity (C&S) × 2 sites, urinalysis (UA), blood culture × 2

Results

White blood cell (WBC): 15.5
Platelets (PLT): 265
Normal chemistry.

Lipase and amylase are normal.
Urinalysis (UA): WBC: 10–25, UA: Bacteria + 1

Procedures

- *Portable chest X-ray (CXR)*: Normal; no acute distress (NAD); normal bowel gas.
- Flat and upright abdominal X-ray.
- *Computed tomography (CT)*: Abdomen and pelvis, PO and IV contrast at 07:30 am.

DISPOSITION (12:20 PM)

Discharge Vitals

BP: 109/70 mm Hg, HR: 123 beats/minute, RR: 24 breaths/minute, *T*: 102°F, and SaO_2: 96%. Pain: 4/10.

Impression

Diverticulitis: LLQ (radiates to groin), leukocytosis and fever.

For most definitive diagnosis: CT

Computerized Tomography

- Diverticula, thickened colonic wall greater than 4 mm.
- Inflammation within pericolic fat more or less.
- Collection of contrast material or fluid.

Differential Diagnosis

- *Sepsis*: Fever, leukocytosis, HR greater than 90 beats/minute, RR greater than 20 breaths/minute, WBC Count less than 4 thousand or greater than 12 thousand is a sign of sepsis.
- *Acute urinary tract infection*: Dysuria, urinary frequency, pyuria, hematuria, bacteriuria, foul and cloudy urine.
- *Acute pyelonephritis*: Fever, dysuria, vomiting, flank pain, U/A: WBC, white cell casts.
- *Inflammatory bowel syndrome*: Abdominal pain, vomiting and diarrhea, rectal bleeding, internal cramps/muscle spasms in pelvic region, weight loss, arthritis, pyoderma gangrenosum and primary sclerosing cholangitis.
- *Acute appendicitis*
- *Sigmoid malignancy*
- *Ovarian cyst*

- *Endometriosis*
- *Pelvic inflammatory disease*

Treatment

One of the following course of treatment is followed.

- *Antibiotics*: Ciprofloxacin and metronidazole
- Ampicillin or sulbactam and piperacillin or tazobactam
- Gentamicin and cefotetan or cefoxitin

Mild Disease

Oral antibiotics: Amoxicillin/Clavulanic acid (Augmentin)

DISCUSSION

True Diverticulum

Sac-like herniation of entire bowel wall.

Pseudodiverticulum

- Mucosa protrusion through muscularis propria of colon is the most common.
- *Protrusion point*: Nutrient artery (vasa recti) penetrates through muscularis propria.
- Break in integrity of colonic wall.
- *Common Site*: Sigmoid colon, 1/20: pancolonic diverticula.
- *Cause*: The main cause is higher amplitude contractions and constipated, high-fat content stool within sigmoid lumen.

DIVERTICULITIS

- Inflammation of diverticulum
- Retention of particulate material within diverticular sac and fecalith formation.
- Compression or erosion of vasa recti causes bleeding indicates perforation.

Acute Uncomplicated Diverticulitis

Presentation: Fever, LLQ abdominal pain, anorexia/obstipation, generalized peritonitis (diverticular perforation)

Examination: Abdominal distension and signs of localized or generalized peritonitis

Laboratory: Leukocytosis.

Diagnosis

Computerized Tomography

- Sigmoid diverticula, thickened colonic wall is greater than 4 mm.
- Inflammation is within pericolic fat.

Contraindications for Testing in Acute Diverticulitis

- Barium enema or colonoscopy in acute setting for fear of perforation and pertonitis
- Perform 6 weeks after diverticular disease to rule out sigmoid malignancy.

Complicated Diverticulitis

- *Presentation*: Abscess > perforation > stricture >> fistula
- *Perforation staging*: Hinchey classification system, predict outcome of postoperative management.
- *Fistula formation*: Cutaneous, vaginal, vesicle fistulae, stool passage through skin or vagina, air in urinary stream (pneumaturia).
- *Colovaginal fistulae*: More common in women with history of hysterectomy.

MANAGEMENT

- Radiographic and hematologic confirmation of inflammation or infection within the colon
- Initially with antibiotic and bowel rest
- Three-fourth of hospitalized acute diverticulitis patients respond to antimicrobial regimen
- Trimethoprim/sulfamethoxazole or ciprofloxacin
  - + Metronidazole: Aerobic gram negative rods + anaerobic bacteria
  - + Ampicillin: If not responding.
- *Single agent*: 3G penicillin, IV piperacillin, oral penicillin/clavulanic acid
- *Duration*: 7–10 days

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Case Study 31: Acute Pancreatitis

CASE HISTORY

A 45-year-old female patient presents with pain that started suddenly in the epigastric region while she was sitting at work. Pain is sharp 8/10 and radiates to the back. Since the onset of the pain, patient has had three episodes of non-bloody, non-bilious vomiting. Patient has never had pain quite like this before, although she states that she has had several episodes of right upper quadrant (RUQ) pain for which she was told that she should eventually have her gallbladder taken out. Past medical history is significant for gastroesophageal reflux disease (GERD) for which she takes omeprazole. Her only past surgery was a tubal ligation after her third pregnancy. She does not smoke, drink alcohol or use any other recreational drugs. On physical examination, patient is afebrile with a blood pressure of 156/95 mm Hg, heart rate 108 beats/minute, respiratory rate 18 breaths/minute, and oxygen (O_2) saturation of 100% on room air. What is the most likely diagnosis? What testing would help to confirm your suspicion?

CASE DISCUSSION

The most likely diagnosis is gallstone pancreatitis. The patient's epigastric abdominal pain radiating to the back is characteristic of pancreatitis. The patient's history of biliary colic suggests gallstones are the cause. Lipase or computed tomography (CT) of abdomen could confirm pancreatitis. Aspartate aminotransferase/alanine aminotransferase (AST/ALT), alkaline phosphatase, electrolytes, complete blood count (CBC) and urinalysis (UA) should also be obtained. Urine pregnancy test in women of child-bearing age should be obtained. Other causes of the patient's abdominal pain that should be considered are biliary colic, cholecystitis, cholangitis, peptic ulcer disease and ulcer perforation.

Acute pancreatitis is an inflammatory disease of the pancreas that is associated with little or no fibrosis of the gland. It has high morbidity and mortality rates contributing to 300,000 hospital admits per year as well as 20,000 deaths. The exact mechanism is unknown and varies with a specific cause. In general, it is thought that there is an activation of digestive zymogens inside acinar cells that leads to injury. In addition, there is activation of inflammatory mediators. If severe, this can lead to systemic inflammatory response syndrome (SIRS) and multiorgan failure or local necrosis of the pancreas.

Typical presentation includes abdominal pain in 95% of cases. The pain is most likely to have a sudden severe onset that is either located

in the epigastric region, RUQ, or less commonly the left upper quadrant (LUQ). The pain has a tendency to radiate toward the back due to the retroperitoneal positioning of the pancreas. The quality is usually boring and deep, alleviated by resting in the fetal position and exacerbated by eating and drinking alcohol. In 90% of cases, there are associated symptoms of nausea and vomiting. Often patients feel restless and agitated. In cases of biliary pancreatitis, the exception is that the pain may come on more gradually and be more localized to the RUQ.

The physical examination depends on the severity of the attack. Mild disease will cause mild abdominal tenderness whereas severe disease will cause severe tenderness and guarding in the upper abdomen. Generally, no rebound tenderness is present. In terms of vitals, patients are usually tachycardic. Mild hypotension may be present due to sequestration of fluid in the pancreatic bed. Low-grade fevers are present without evidence of infection in 60% of cases. Respirations are generally rapid and shallow. Signs suggestive of severe disease include Gray Turner's sign and Cullen's sign. Gray Turner's sign consists of ecchymoses in the flanks that are indicative of hemorrhage from hemorrhagic pancreatitis. Cullen's sign is ecchymoses in the periumbilical region that is similarly suggestive of intra-abdominal hemorrhage. Other associated findings that are suggestive of peripancreatic spread of inflammation include generalized ileus, signs of pleural effusion, subcutaneous fat necrosis and jaundice.

Expected laboratory findings include leukocytosis, mild hyperglycemia, mild increase in AST/ALT from alcoholic pancreatitis, large increase in AST/ALT from biliary causes. More importantly, serum lipase has a high sensitivity of 90% and high specificity when elevated up to two times normal. Serum amylase is sensitive but not specific for pancreatitis unless three times normal. Other pancreatic enzymes can be measured but generally not done clinically.

Abdominal CT is the most accurate for diagnosing pancreatitis and identifying its localized effects. CT abdomen should be done when you are uncertain or the diagnosis, suspect complications, or in cases of mild to moderate disease that do not improve over several days. Intravenous (IV) and oral contrast should be used. Findings on imaging will include pancreatomegaly, peripancreatic streakiness or "dirty fat", necrotic areas that fail to enhance. CT severity index may be used as an aid in prognosis. Other methods of imaging that may be used are abdominal X-ray, ultrasound, and magnetic resonance cholangiopancreatography (MRCP). Abdominal X-ray is often used to rule out perforation. Findings include dilated loops or air-fluid levels secondary to paralytic ileus, sentinel loop, colon cut-off sign, widening of the C-loop, and calcifications of the gallbladder. Ultrasound may provide evidence of the cause of

pancreatitis and may aid in ruling out gallstones. It is often difficult to identify the pancreas due to overlying intestinal gas or fat. If able to visualize, a hypoechoic and enlarged pancreas may be present. MRCP is useful for selecting patient populations: pregnancy, contrast allergy and renal insufficiency. It is most accurate for visualizing bile and pancreatic ducts and pancreatic fluid collections. This test may be most useful while awaiting endoscopic retrograde cholangiopancreatography (ERCP) for definitive management.

There are many causes of pancreatitis. It is important to identify the cause in order to direct the present therapy and prevent future recurrences. The most common cause overall is gallstones making up to 40%. Combined, gallstones and alcoholism can cause 75% of cases. In 15% of cases, the cause can never be identified. For alcoholic pancreatitis, it requires more than 8 alcoholic drinks per day for more than 5 years. Smoking is an important cofactor. Frequently alcoholics develop chronic pancreatitis. Hypertriglyceridemia accounts for 2% of the cases. Triglyceride levels greater than 1,000% are suggestive, but levels greater than 2,000% are diagnostic. Levels should be measured early. If found to be greater than 10,000%, plasmapheresis may be required. It has been suggested that mumps, coxsackie and mycoplasma infections may cause pancreatitis because titers have been shown to be elevated during an attack, but none have been isolated in the pancreas. ERCP causes 2% of cases. Mild to moderate elevations in amylase and lipase occur after ERCP regardless, so it is unnecessary to measure levels immediately after the procedure.

Patients with pancreatitis are most likely going to be admitted except in some cases of chronic pancreatitis. However, a decision has to be made on whether to admit these patients to the intensive care unit (ICU). This is where the severity of the criteria can be useful. Ranson's CT severity index, acute physiology and chronic health evaluation II (APACHE II) are the most commonly used. APACHE II generally has the best predictive value, but the criteria involves more testing. Please see Box 1 and Table 1 for these criteria. Any signs of organ failure warrant admission to the ICU. For this to happen, coordination must take place between the ICU team, gastrointestinal (GI) team, surgery and infectious disease. First line therapy is supportive. Aggressive fluid hydration is required to prevent acute tubular necrosis (ATN) and lessen pancreatic necrosis for 48 hours. Glucose should be monitored because elevations may occur even without history of diabetes. Analgesia is essential for patient comfort. Historically, meperidine 50–100 mg q 6 hours has been used, but long-term build-up of toxic metabolites has caused this drug to fall out of favor. It was once thought that morphine use would worsen pancreatitis due to sphincter

Box 1: Ranson's criteria

Ranson's Criteria on Admission:

- Age over 55 years
- A white blood cell count of > 16,000/µL
- Blood glucose > 11 mmol/L (> 200 mg/dL)
- Serum LDH > 350 IU/L
- Serum AST > 250 IU/L

Ranson's Criteria 48 hours of Admission:

- Fall in hematocrit by more than 10%
- Fluid sequestration of > 6 L
- Hypocalcemia (serum calcium < 2.0 mmol/L (< 8.0 mg/L))
- Hypoxemia (PO_2 < 60 mm Hg)
- Increase in BUN to > 1.98 mmol/L (> 5 mg/dL) after IV fluid hydration
- Base deficit of > 4 mmol/L

The prognostic implications of Ranson's criteria are as follows:

- Score 0–2: 2% mortality
- Score 3–4: 15% mortality
- Score 5–6: 40% mortality
- Score 7–8: 100% mortality

Table 1: CT Severity index

Prognostic Indicator	*Points*
Pancreatic inflammation	
Normal pancreas	0
Focal or diffuse enlargement of the pancreas	1
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2
Single, ill-defined fluid collection of phlegmon	3
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4
Pancreatic necrosis	
None	0
≤ 30%	2
> 30–50%	4
> 50%	6

of Oddi contraction, but this is now thought to be more theoretical. Morphine is frequently used for pain control. Patients should be nil per os (NPO) for bowel and pancreatic rest. Diet should be advanced slowly when there is no longer pain, nausea, vomiting or distention. If patient is expected to be NPO for more than a few days, then parenteral nutrition

should be considered with total parenteral nutrition (TPN) or nasojejunal (NJ) tube. Nasogastric tubes are no longer used because randomized trials did not show faster times to per os (PO) intake. Vitals should be frequently monitored.

Directed therapy depends on the cause of pancreatitis. For patients with gallstone pancreatitis, ERCP and sphincterotomy may be considered in conjunction with GI consult. This should be urgently done if cholangitis is suspected. Cholecystectomy should be done at some point during the hospitalization. Timing is controversial but typically done between 48–72 hours or after 72 hours. Intraoperative cholangiogram should be done to rule out the presence of stones in the common bile duct if ERCP has not already been done.

Many complications can occur as a result of pancreatitis. It is important to be aware of these in order to both prevent and rapidly treat them. The most common complication, which occurs in 10% of patients is a pancreatic pseudocyst, which is defined as a collection of pancreatic fluid surrounded by a nonepithelialized wall of granulation tissue. Typical presentation includes a history of pancreatitis with now worsening pain, distention, and difficulty eating. Drainage is required whether it is done by CT-guided endoscopy or surgery. Regardless of the mode of procedure the treatment involves making a connection between the cyst and one of three places: stomach, duodenum or jejunum. Surgical intervention is done when the cyst lasts greater than 6 weeks and/or is greater than 5 cm. A severe complication is pancreatic abscess. This typically occurs 2–6 weeks after the initial attack. Patients will become septic. CT scan is necessary to diagnose and treatment is drainage. Mortality rate is high. Other complications include: hypocalcemia, disseminated fat necrosis, *acute respiratory distress syndrome* (ARDS), renal insufficiency, sterile pancreatic necrosis, infected pancreatic necrosis in the first few hours or days, ascending cholangitis, pancreatic abscess, abdominal compartment syndrome, splenic artery or gastroduodenal artery pseudoaneurysm, pancreatic fistula, pancreatic ascites, splenic vein thrombosis, cardiovascular shock with hypovolemia and hemorrhagic pancreatitis.

In summary, the most common symptoms are abdominal pain, nausea and vomiting. Lipase has the highest sensitivity and specificity. CT is the most sensitive imaging, but it is not always needed for diagnosis. The most common causes are gallstones and alcoholism. It is important to assess the need for ICU admission. The mainstay of treatment is supportive therapy with aggressive fluid resuscitation, NPO and analgesia.

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Case Study 32: Acute Cholecystitis

CASE HISTORY

A 26-year-old Caucasian female is presented to the emergency department with a chief complaint of right upper quadrant pain, associated with nausea and vomiting, after a night at the pizza parlor with her friends. She is obese with a body mass index (BMI) of 35 kg/m^2. Her vital signs are temperature 37.7°C, pulse rate 120 beats/minute and score on pain scale is 9/10. On examination, there is tenderness in the right upper quadrant. Murphy's sign cannot be elicited because of severe pain in that area. After conducting a complete blood count (CBC), white blood cell (WBC) count is 12,000/µL with no shift, serum lipase is within normal limits and liver enzymes are slightly elevated. A gallbladder ultrasound is done by the doctor, which is shown below, shows two gallstones and thickening of the wall to the gallbladder (Fig. 1). Upon seeing the ultrasound image the nurse exclaimed, "That's where I left my marbles!"

DIFFERENTIAL DIAGNOSES

Differential diagnoses of cholecystitis is shown in Figure 2.

TREATMENT

Watchful waiting is the best treatment option for most patients with asymptomatic gallstones. Patients with cholecystitis should have a

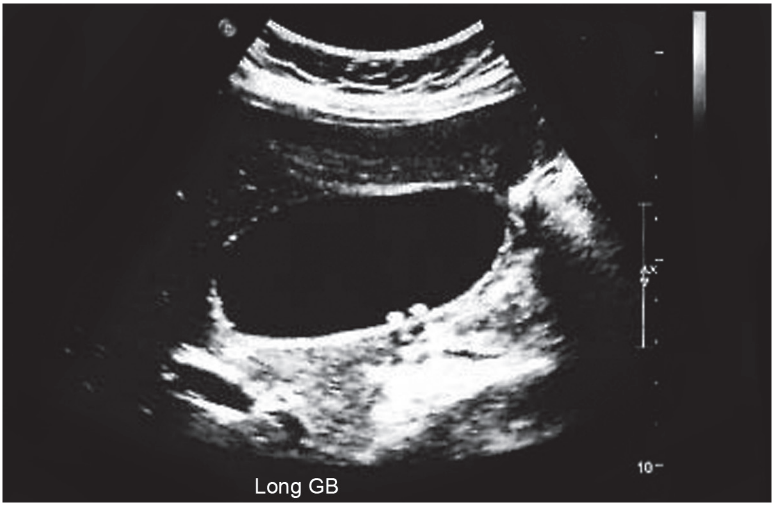

Fig. 1: Bedside ultrasound in the ER done by the author showing gallstones

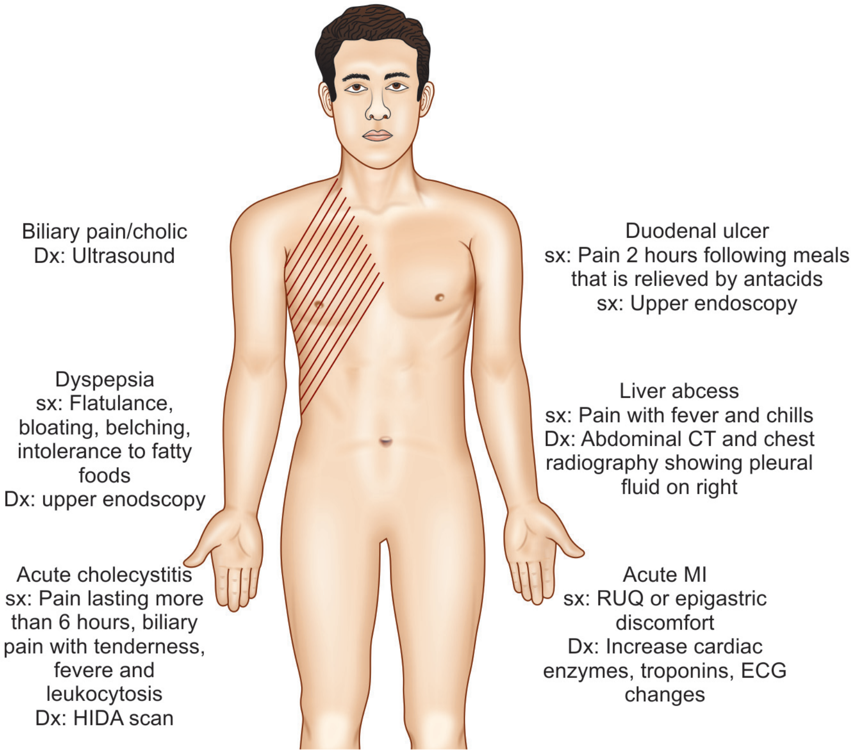

Fig. 2: Differential diagnosis of acute cholecystitis (by symptoms and diagnostic tests)
Source: Modified from Gastrointestinal Emergencies, 2nd Edition. Diverticular Disease. Blackwell Publishing Ltd 2009.

laparoscopic cholecystectomy in the early course of treatment. According to Bellows, Berger and Crass (2005) gallstone pancreatitis requires a laparoscopic cholecystectomy.[1] Oral dissolution therapy is appropriate for those who are not fit for surgery [elderly, at risk, heart conditions, past medical history (PMH) including stroke]. Selection criteria is based on three main characteristics: age of patient, condition of gallblader, and characteristics of the stone.[2]

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CHAPTER

4

Pelvic and Urogenital

Case Study 33: Testicular Torsion

CASE HISTORY

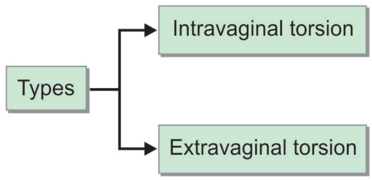
Patient is a 14-year-old male who is presented to emergency department (ED) with a chief complaint of pain in the right testis since yesterday which continues to worsen. The patient is now experiencing difficulty in walking. He reports that he had similar pain in the past, but this time it is more severe. He denies any history of trauma, but states that he was exercising strenuously in the gym a few nights ago. Patient denies fever and history of sexually transmitted infections (STIs). His vital signs are temperature 97.4 F, pulse rate 90 beats/minute, blood pressure 100/62 mm Hg, and pulse oximetry 98% on room air. Physical examination reveals an enlarged, erythematous, edematous and extremely tender scrotum on the right side. The cremasteric reflex is absent. Doppler ultrasound is ordered immediately, and it reveals swelling and lack of flow. Radionuclide testicular scan with technetium-99 pertechnetate (also known as Tc-99m) demonstrates decreased vascularity, venous thrombosis, and tissue edema. Patient has a presumed testicular torsion and an immediate urologic consultation is obtained for definitive management.[1]

DISCUSSION

Each year testicular torsion affects one in 4,000 males younger than 25 years. All prepubertal and young adult males with acute scrotal pain should be considered to have tersticular torsion until proven otherwise.

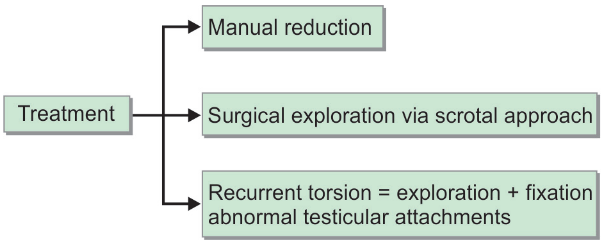
Based on the presentation, the patient likely has a testicular torsion. Given the presence of right testicular pain several other diagnostic considerations may be made, but torsion is a “do not miss” diagnosis because over time the testis will no longer be viable due to a disruption in blood flow.[2] The differential includes epididymo-orchitis, incarcerated inguinal hernia, hematoma either traumatic or idiopathic, acute hydrocele, testicular tumor, epididymitis, orchitis, spermatocele, hydrocele, varicocele, or torsion of the appendix testis.

Flow chart 1: Types of testicular torsion



Source: Badar M Zaheer, MD

Flow chart 2: Treatment of testicular torsion



Source: Badar M Zaheer, MD

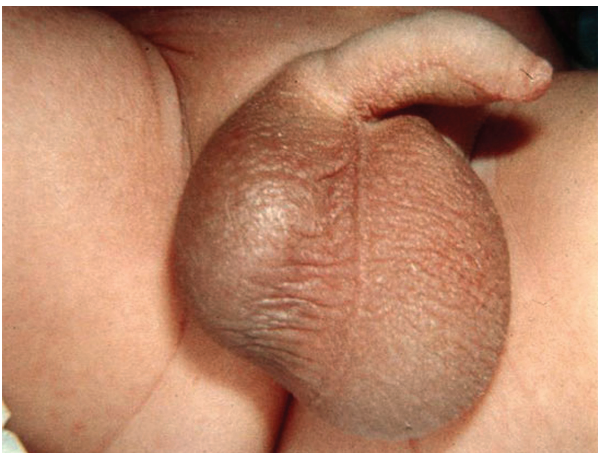


Fig. 1: Torsion of testicel—one testis is appearing less descended than the other
Source: http://www.sciencephoto.com/media/146577/view

Testicular torsion is defined as twisting of testis and spermatic cord causing acute ischemia (Fig. 1). The most common anomaly is high insertion of tunica vaginalis on the spermatic cord causing increased testicular motility bilateral in 80% of patients (Flow chart 1). Presentation includes pain, swelling, and absence of cremasteric reflex.[3] Absence of ipsilateral cremasteric reflex is the most accurate sign of testicular torsion. Blue dot sign (a tender nodule with blue discoloration located at the upper pole of the testis) may also be seen and used for diagnosis. One testis may appear less descended than the other (Fig. 1) Ultrasound with Doppler flow may confirm diagnosis (Fig. 2). Testicular torsion is a true

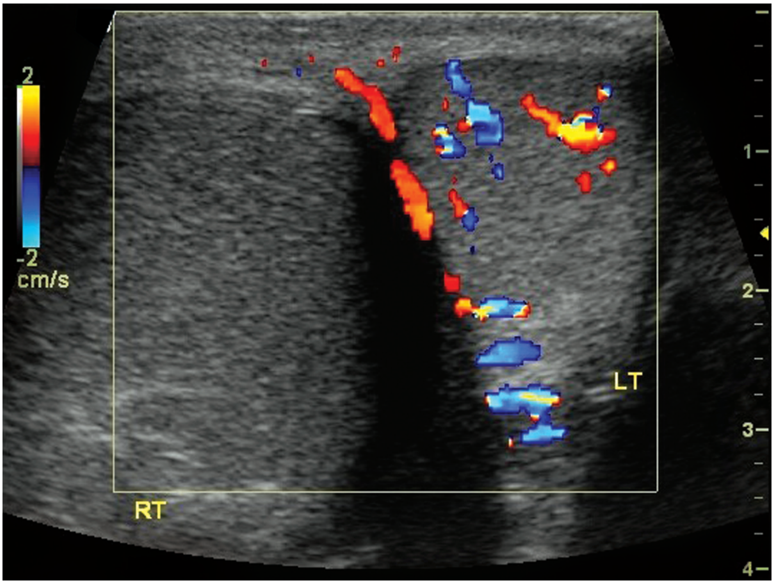


Fig. 2: Ultrasound with Doppler flow showing testicular torsion
Source: http://radiology.casereports.net/index.php/rcr/article/viewArticle/299/732

urologic emergency (Flow chart 2). If the physical exam is imperfect or inconclusive, quick imaging studies like ultrasonography and nuclear scans, are very useful when the testicular torsion has to be ruled out or ruled in. Very early urology consult is mandatory for detorsion and orchipexy or possible orchiectomy. If urology consult in the ER is unavailable, a quick transfer to urologists with sufficient analgesic medication

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CHAPTER

5

Musculoskeletal

Case Study 34: Septic Arthritis

CASE HISTORY

A 40-year-old female is presented to the emergency department (ED) with left knee pain for the last 3 days. Patient noticed the pain when she woke up 3 days ago. Pain has been worsening since then. She is having difficulty in ambulating due to excruciating pain. She was having no fever at home. No history of sexually transmitted infections is present. No history of gout or arthritis is there. Patient is afebrile with unremarkable vitals. Physical examination reveals a tender, erythematous, warm joint on the left knee. Ambulation is extremely limited due to pain. Due to concern for septic arthritis, the joint is tapped. Results of the tap include approximately 5 cc of yellow-green fluid. White blood cell (WBC) count is greater than 100,000/μL with initial gram stain inconclusive. Fluid is sent for culture, and blood cultures are also drawn at this time. Intravenous (IV) antibiotics are started. Orthopedic consult is obtained for possible arthroscopy.

DISCUSSION

Septic arthritis is a form of infectious arthritis in which various microorganisms invade the joint.[1] Risk increases with prosthetic joints as well as rheumatoid arthritis (RA) and *systemic lupus erythematosus* (SLE). Classification is either gonococcal or nongonococcal. The most common pathogen is *Neisseria gonorrhoea* among young, sexually active individuals. However, overall the most common cause is *Staphylococcus aureus*. The knee is the most commonly involved joint.

Septic arthritis is a medical emergency because bacterial invasion can lead to destruction of the joint.[2] The destruction results from initial damage to articular cartilage. The pathophysiology starts with destruction, followed by pannus formation and subsequently cartilage erosion. Severe damage may occur in a little as 3 days. Morbidity from dysfunction of the

joint can manifest as decreased range of motion or chronic pain. Other complications include dysfunctional joints, osteomyelitis and sepsis. Prognostic signs for poor outcomes include age greater than 60 years, infection in hip or shoulder joints, underlying RA, positive synovial fluid cultures after 7 days of appropriate antibiotics and delay of 7 days in starting therapy. Mortality rate is low for *N gonorrhoea*, but may be as high as 50% in cases where *S aureus* is the causative agent.

History should determine acute onset of joint pain, or superimposed acute on chronic symptoms, history of trauma, which joints are involved, presence of extra-articular symptoms and use of IV drugs. Patients should be questioned regarding exposure to sexually transmitted infections, exposure to ticks, and immunocompromised risk factors. Physical examination should assess for signs of erythema, swelling, warmth and tenderness. Effusions are generally present when the joint is infected. Assess active and passive range of motion which will usually be markedly limited. Differential includes Lyme disease, prosthetic joint infection as well as reactive and tuberculous arthritis.

Synovial fluid should be obtained in all suspect cases. Even patients with a history of gout warrant synovial fluid analysis. Septic joints will generally have a lack of crystals, pus like synovial fluid, gram stain may be positive, WBC will exceed 50,000/μL with the majority of polymorphonuclear leukocytes. Polymerase chain reaction (PCR) may further assess for specific viral or bacterial causes. PCR is especially helpful in the setting of patients who have recently been given antibiotics that would otherwise influence the culture. Blood cultures should also be obtained to look for bacteremic origin, like swabbing, rectum, cervix, urethra, or pharynx of confirm possible gonococcal infection X-ray may assess for osteomyelitis (Fig. 1). Ultrasound may confirm effusions in distorted joints.

TREATMENT

Treatment should include admission, pain control, consultation and antibiotic therapy. Length of antibiotic course depends on the pathogen. In general, at least 2 weeks will be required. Some cases with complications, such as osteomyelitis, will require PORT-A-CATH placement and several months of antibiotics. Start empirically and tailor to both the hospital resistance patterns and the cultures. Consider starting with a combination of penicillin and gentamicin or a later generation cephalosporin (Fig. 2).

PITFALLS OF MISSING SEPTIC ARTHRITIS

All patients with a presentation of monoarticular joint pain should be assumed to have septic arthritis, until proven otherwise. Severe medical

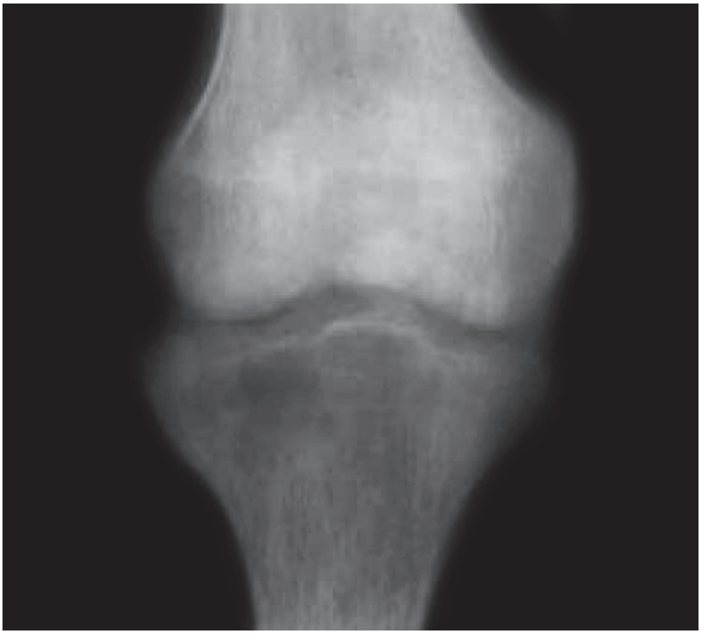

Fig. 1: X-ray shows septic arthritis[2]
Source: http://emedicine.medscape.com/article/395381-overview

Fig. 2: Aspiration of keen joint for synovial fluid analysis
Source: http://reference.medscape.com/features/slideshow/arthro-practice

malpractice may result from a missed case. This is because of delays in treatment that can result in severe morbidity as well as mortality. Long-term disability is a strong motivator for taking legal action. Consider

a young man with a missed diagnosis, the misery and long-term disability after knee surgery. Regardless of why the diagnosis was missed, the patient required a skin graft, and eventual above the knee amputation. This story emphasizes why it is always important to consider this diagnosis.

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Case Study 35: Acromioclavicular Joint Separation

ACROMIOCLAVICULAR JOINT INJURIES

A 23-year-old man is presented to the emergency room with shoulder pain after falling on his left shoulder (Fig. 1). On physical examination, his vital signs are temperature 98.5°F, pulse rate 84 beats/minute, respiratory rate 16 breaths/minute, and blood pressure 120/80 mm Hg. Examination shows tenderness of the left lateral clavicular area. He is unable to abduct his shoulder, internal rotation is restricted and painful, but he can touch his right shoulder with his left hand. Radial and brachial pulses are palpable and there is no evidence of neurovascular compromise.

X-ray of the shoulder is shown below as Figure 2.

The diagnosis is based on the physical examination and findings of an X-ray of AC joint separation due to a torn coracoclavicular ligament.

FEATURES OF ACROMIOCLAVICULAR JOINT INJURIES

Acromioclavicular (AC) joint injuries are classified as Type I, Type II, or type III based on the severity of the injury and the X-ray findings (Flow chart 1). A Type I injury is a strain or partial tear, which will have normal X-ray findings. Type II injuries result in a disruption in the AC

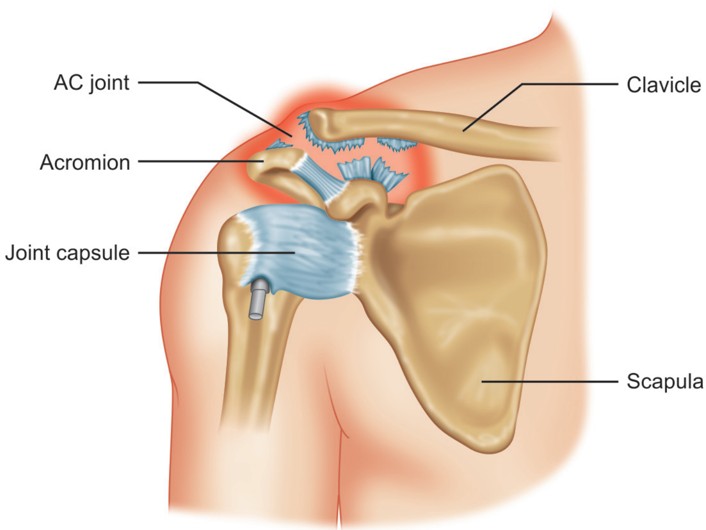


Fig. 1: Anatomy of AC joint

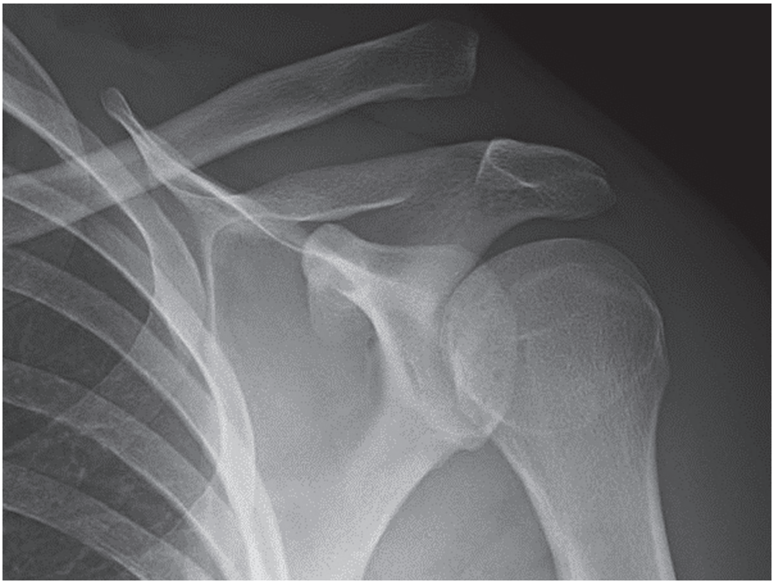
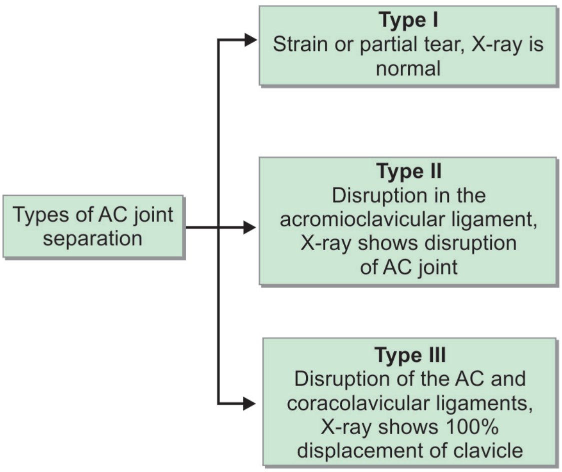


Fig. 2: X-ray of the shoulder showing AC joint separation

Flow chart 1: AC joint separation types



ligament. If X-ray shows 25–50% elevation of the clavicle above the acromial process, then Types I and II will have a widening of the coracoclavicular space between 1.1 mm and 1.3 mm. Type III injuries are associated with a complete disruption of the AC and coracoclavicular ligaments and are identified on X-ray by a 100% superior displacement of the clavicle and a widening of the coracoclavicular space of greater than 5 mm.

TREATMENT

Treatment of Type I and II injuries is done conservatively with rest, ice, analgesics, and arm immobilization with a sling for 1–3 weeks. Range of motion and strengthening exercises are started once the patient gets better and is pain free. For Type III injuries, treatment approach is controversial. Many prefer conservative treatments similar to that of Types I and II, although some physicians opt for surgical repair. More severe injuries are managed surgically.

Case Study 36: Glenohumeral Joint Dislocation

CASE HISTORY

A 19-year-old baseball player is presented to the emergency department complaining to right shoulder pain. The injury occurred during a baseball game when he slid head first into second base with his arms outstretched.

On physical examination, his vital signs are stable with a temperature of 98.7°F, pulse rate 74 beats/minute, respiratory rate 16 breaths minute and blood pressure 116/70 mm Hg. He holds his arm in a slightly-flexed position, supported by the left hand. The shoulder has a "squared off" appearance and he complains of severe pain on examination. He refuses to move the extremity.

X-ray of the shoulder is shown in Figure 1.

CLINICAL FEATURES

Glenohumeral joint dislocation is the most common type of joint dislocation (Flow chart 1). 95–97% of these injuries are anterior dislocations caused by indirect force on the joint capsule with the arm abducted, extended, and externally rotated. The patient is presented

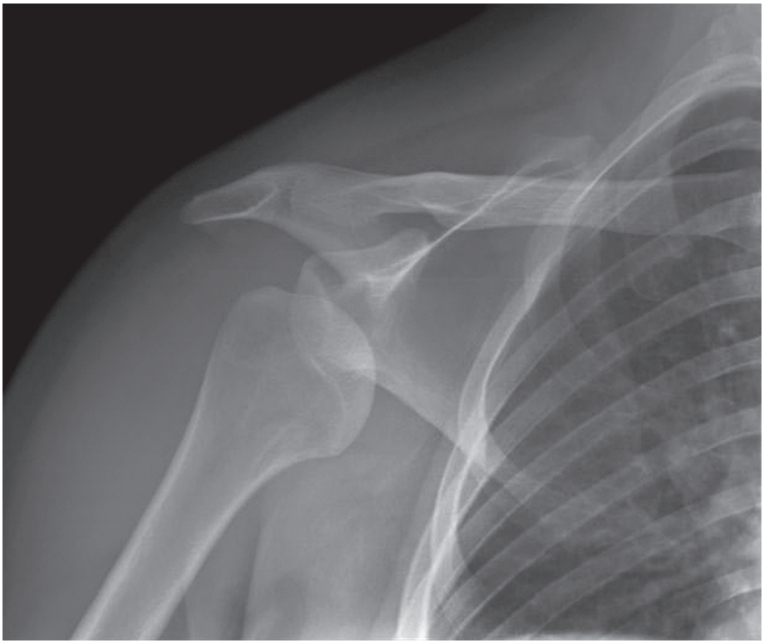
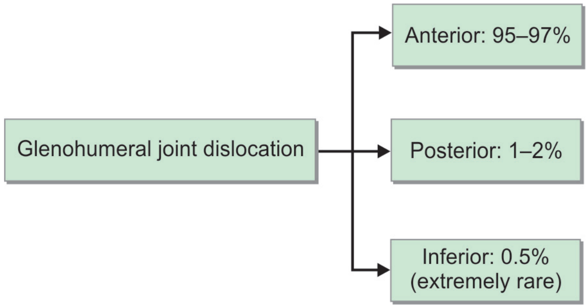


Fig. 1: Shoulder X-Ray
Source: Badar M Zaheer, MD

Flow chart 1: Types of Shoulder Dislocation



Source: Badar M Zaheer, MD

with severe pain and holds the joint in a slightly abducted and externally rotated position, supported by the other arm. On visual inspection, the shoulder appears “squared off” and shortened. The most common complication is axillary nerve injury. Function can be assessed by pinprick sensation over the skin of the lateral shoulder.

DIAGNOSIS

Diagnosis is made by physical examination combined with anteroposterior (AP) and lateral scapular “Y” or axillary X-ray views. The X-ray can also often identify concomitant fractures, although these require no additional treatment. Common associated injuries include compression fractures of the humeral head, anterior glenoid rim fractures, greater tuberosity avulsion fracture, and rotator cuff tears.

TREATMENT

Anterior glenohumeral joint dislocations are treated with reduction followed by immobilization of the joint. There are a number of techniques that can be used to reduce the dislocation, the most common of which is the modified Hippocratic technique.

Closed reduction with IV sedation and muscle relaxation.

1. Modified Hippocratic technique.
2. *Stimson technique*: While patient lies prone with arm hanging over the edge of the table, hang a 5 lb weight on wrist for 15–20 minutes applied.
3. *Traction—counter-traction technique*: An assistant stabilizes the torso with a folded sheet wrapped around the patients chest while the physician applies gentle steady traction. See Fig. 4.

Obtain postreduction X-rays and check for neurovascular status. Put shoulder in sling for 3 weeks followed by physical therapy. Immediate orthopedic referral for failed/unstable closed reductions.

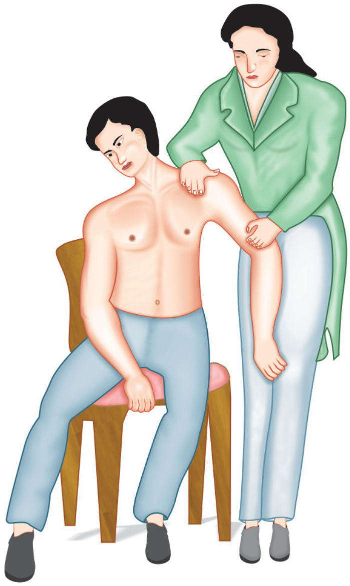


Fig. 2: Patient's treatment with Hippocratic technique

Fig. 3: Patient's treatment with Stimson's technique

Source: Available online from http://www.eorthopod.com/sites/default/files/images/shoulder_dislocation_treatment01.jpg

OTHER COMMON TREATMENT TECHNIQUES

In the Stimson's technique, the patient is placed prone on a gurney with the dislocated shoulder hanging off the side A 10-pound weight is hung

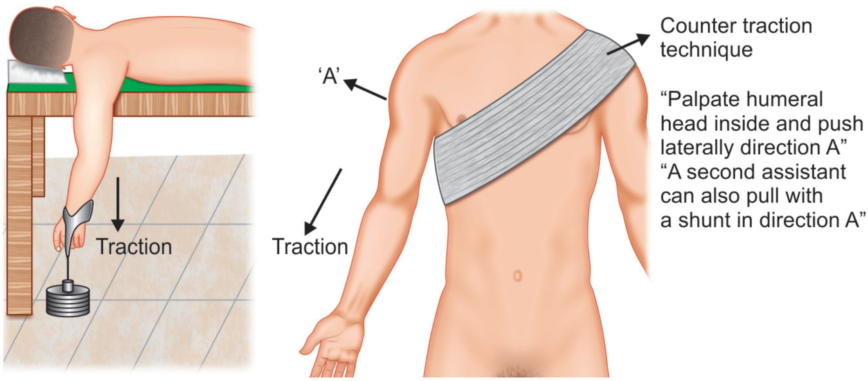


Fig. 4: Reduction of anterior dislocation. Traction and counter-traction technique. Assi stant stabilizes the torso with a folded sheet wrapped across the chest while the physicians applies gentle steady traction

off the wrist (Fig. 3) If muscle relaxation is obtained, reduction can occur in 30 minutes.

Aronen's technique is used for recurrent dislocations. This technique is useful because it can be taught to the patients for self-reduction of an injury. In this method, the patient is seated on a gurney with the ipsilateral leg and knee in flexion. The patient is then told to clasp his hands behind the knee and relax the shoulder muscles. Countertraction is applied by the patient's own body weight and his own paraspinous muscles. Taping the patient's hands together can help reduction.

POSTERIOR DISLOCATIONS

These account for approximately 1–2% of glenohumeral dislocations. They occur with convulsive seizures, direct trauma to the anterior shoulder, or falls on an outstretched arm. The patient presents with the arms held across the chest in adduction and internal rotation. On active range of motion examination, abduction is severely compromised and external rotation is completely blocked. On inspection, the anterior shoulder appears flat with a prominent coracoid process and the posterior aspect appears full. Anteroposterior X-ray shows a loss of the elliptical half-moon-shape overlap seen on normal films. The humeral head has a "light bulb" or "drumstick" appearance. The diagnosis can be confirmed with scapular Y-view of lateral views. Reduction is done on a supine patient by applying axial traction along the long axis of the humerus, anterior directed pressure on the posterior humeral head, and some external rotation.

INFERIOR DISLOCATIONS

The injuries are very rare and make up only about 0.5% of shoulder dislocations. The mechanism of injury is usually a forceful hyperabduction or an inferior directed axial load on the abducted shoulder. The patient will present with the arm-locked overhead. Reduction is completed with an upward and outward force in line with the humerus.

ADDITIONAL READING

1. Anterior Shoulder Dislocation http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2752231/

Case Study 37: Low Back Pain

CASE HISTORY

A 52-year-old male presents with low back pain of 4 months. Patient reports that he has had low-back pain problems for "years", but over the last 4 months it has been worsening to now include left-sided weakness. It is now to the point where the patient is having difficulty staying still for any amount of time Only standing in certain positions alleviates the pain. Patient has been taking over-the-counter (OTC) NSAIDs without relief. He occasionally has shooting and tingling pain down to his foot. He decided to come in today because he just can not take the pain anymore. He has a 54 pack-year history of smoking, drinks 6–12 beers per day, and denies recreational drugs. Patient is afebrile and normotensive. On physical examination, he does not have spinal tenderness, but is positive for paraspinal tenderness. Cranial nerves are intact. Strength is 5/5 in upper extremity flexors and extensors, but it is difficult to assess strength in the lower extremities due to pain. He has a loss of muscle bulk in the left anterior thigh. The sensation to pinprick is intact throughout. Reflexes are symmetrical. He has no cerebellar signs. Gait is difficult to assess due to pain. Straight leg raise test is positive on the left. Toe is downgoing. Rectal tone is intact. What should be taken into consideration when deciding if this patient should be imaging? What should be done and how quickly?

Should Imaging be Done for this Patient?

- Usually conservative management for the first 4–6 weeks is sufficient.
- *Acute Concerns*: Epidural abscess, epidural compression syndrome, malignancy, spinal stenosis an, back pain in children. Always think of *abdominal aortic aneurysm* (AAA) in hypotensive and, elderly patients.

Indications for Plain Films

- Patient's Age should be less than 18 years or more than 50 years.
- Any history of malignancy or unexplained weight loss.
- Any history of fever, immunocompromised status, or IV drug use.
- Recent trauma other than simple lifting.
- Progressive neurologic deficits or other findings consistent with cauda equina syndrome.
- Prolonged duration of symptoms beyond 4–6 weeks.

Additional views are given if spondylolysis or spondylolisthesis suspected.

Look for Red Flag Signs

History

Patient should be in age group of under 18 years or over 50 years, with pain lasting for more than 6 weeks, history of cardiac arrest, fevers or/chills, night sweats or unexplained weight loss, recurrent bacterial infection, unremitting pain, night pain, IV drug users, major trauma and, minor trauma in the elderly patients.

Physical Examination

Below mentioned points should be checked during physical examination: Fever, writhing in pain, bowel or bladder incontinence, saddle anesthesia, decreased or absent anal sphincter tone, perianal sensory loss, severe or progressive neurological defect an, major motor weakness.

Computed Tomography versus Magnetic Resonance Imaging

Computed tomography (CT) scan is done for evaluating the spinal canal and risk for impingement. For cauda equina, spinal infection, and malignancy, magnetic resonance imaging (MRI) scan is useful. MRI scan has to be emergently done if neurologic findings are acute or progressive.

FOLLOW-UP CASE OUTCOME

Given patient's age, recent weight loss, and history of smoking, a lumbosacral X-ray was ordered, but found to be inconclusive. CT scan of lumbosacral spine was ordered and showed evidence of multiple areas of disk bulging, but no fractures or signs of metastasis. Given history of acute and chronic weakness, emergent MRI was considered. However, the patient was reassessed after receiving pain medication and it was determined that he did not have weakness on physical examination Patient was instructed to follow-up with an MRI scan as an outpatient, as well as with neurosurgery for further evaluation.[1]

ASSESSMENT OF BACK PAIN

It is important to do a thorough neurovascular examination including deep tendon reflexes, sensation, and muscle strength. It is also important to assess peripheral pulses and palpate the abdomen for masses or organomegaly. Lower extremities should also be assessed for flexibility and the spine should be examined for posture, stance, gait, and straight leg raise.

MANAGEMENT

No-surgical management is focused on pain control, bracing and rehabilitation. This usually requires extensive inpatient physical therapy,

occupational therapy, and recreational therapy.[2,3] Never miss a spinal epidural abscess for it can lead to a devastating and irreversible outcome. Always keep this diagnosis in mind for every backache complaint.

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Case Study 38: Headache

"Holding on to anger, resentment and hurt only gives you tense muscles, a headache and a sore jaw from clenching your teeth. Forgiveness gives you back the laughter and the lightness in your life."

—John Lunden

CASE HISTORY

Patient is a 46-year-old female who presents with headache of 4 days. Patient states that she woke up in the morning with a headache 4 days ago. Since then the headache has been waxing and waning but has never been completely resolved. She has never had headaches in the past, and describes this headache as the worst of her life. Ibuprofen which she also takes for her osteoarthritis has not been alleviating her pain. For the past week, the patient has generally been feeling unwell without specific symptoms. She does have associated photophobia and nausea but no vomiting. The patient states that her neck is stiff although she has full neck range of motion (ROM). She has no fevers or chills. No intravenous (IV) drug use, no history of immunocompromise, no loss of consciousness (LOC), no head trauma and no numbness, tingling, or weakness were observed. Past history includes hypertension, diabetes type 2 and osteoarthritis for which the patient takes metformin, lisinopril, *hydrochlorothiazide* (HCTZ) and ibuprofen. Surgical history includes tubal ligation and family history of diabetes. Socially, the patient does not smoke, drink, or use other drugs. At baseline, the patient has difficulty getting around her house. On physical examination, patient had temperature 36.8°C, blood pressure 160/94 mm Hg, pulse rate 76 beats/minute, respiratory rate 20 breaths/minute, and oxygen (O_2) saturation 96%. Head, eyes, ears, nose and throat (HEENT) and neck examination were significant for photophobia, but negative for meningeal signs and sinus tenderness. Neurologically, the patient did not have any deficits. Cranial nerves II–XII are intact. Strength is 5/5 in upper and lower extremity flexors and extensors. Sensation is intact to pinprick throughout. Reflexes are 2+ throughout. No cerebellar signs were noticed. She has normal gait.

CASE DISCUSSION

Differential for this patient includes migraine headache, *subarachnoid hemorrhage* (SAH), and viral meningitis. Patient has nausea and photophobia consistent with migraine, but no history of migraines in the past. The positive sudden severe onset warrants consideration of SAH. Viral meningitis is a possibility because the patient had generally

been feeling unwell in addition to some subjective neck stiffness, but did not have any fevers or toxic appearance that would suggest a bacterial meningitis. Computed tomography (CT) and lumbar puncture (LP) were done in a given time frame where CT scan would have a decreased sensitivity. CT scan was unremarkable. *Cerebral spinal fluid* (CSF) analysis clear in appearance. opening pressure was 24 cm H_2O. High lymphocyte count with elevated protein and normal glucose and clearing of red blood cells (RBCs) from Tube 1 to Tube 4. Patient was tranferred to a university hospital for further treatment and speciality case.

HEADACHE DISCUSSION

The initial differential is aimed at determining whether the cause of the headache is primary or secondary. Primary causes include migraine, tension-type headaches, cluster headache, chronic paroxysmal hemicrania, miscellaneous headaches unassociated with structural lesion (idiopathic stabbing, external compression, cold stimulus, benign cough, benign exertional, associated with sexual activity). Secondary causes include head trauma, vascular disorders [*cerebrovascular accident* (CVA), *arteriovenous malformation* (AVM), *cerebral venous thrombosis* (CVT) and SAH], nonvascular intracranial disorder (neoplasm), substance use or withdrawal, infection, metabolic disorders, craniofacial disorder (including cranium, neck, sinuses, etc.) and neuralgias.

Several clinical pearls can be used to determine a likely cause of the headache. Please see Table 1.

Table 1: Findings and considerations of several clinical pearls

Finding	*Consideration*
Thunderclap headache	Subarachnoid hemorrhage (SAH)
Worst headache	SAH, cerebral venous thrombosis (CVT)
Use of space heater	Carbon monoxide
Pregnancy	Eclampsia, CVT
Change in vision	Glaucoma, optic neuritis
Pain with eye movement	Optic neuritis
Fever	Infection (CNS vs systemic)
Double vision	Intracranial mass, idiopathic intracranial hypertension
Ptosis, miosis	Carotid artery dissection
Papilledema	Mass lesion, optic neuritis, pseudotumor
Dilated pupil	Aneurysm compressing third nerve
Age over 50 years	Temporal arteritis, mass lesion, glaucoma

Pearls

Studies show that onset/severity may be the most predictive of a SAH, meaning headaches that initiate suddenly with excruciating pain. According to Godwin and Villa (2001), "In one prospective study, 70% of patients (35/49) presenting with a thunderclap headache has an SAH. Another study prospectively examined all patients presenting with severe headache of sudden onset with no past history of the same. Of 27 patients enrolled, nine had SAH, one had intraventricular hemorrhage, and two had meningitis".[1-3]

Several danger signals may be used to identify the high-risk headaches. Historical danger signals include sudden onset of headache (thunderclap), worst headache of life, headache dramatically different from past headaches, immunocompromise, new onset of headache after the age of 50 years, headache that begins with exertion. Danger of physical findings includes altered mental status, meningeal signs, positive "jolt" test, focal neurologic signs, rash suspicious for spotted fever and meningococcemia.

In 2008, American College of Emergency Physicians (ACEP) created guidelines to better help and determine what evidence was in the literature on the management of headaches. Based on their guidelines, level A is generally accepted with strong evidence. Level B has moderate clinical certainty. Level C has preliminary, inconclusive, or conflicting evidence.

The guidelines argue that there is very little evidence that response to therapy which can be used on a diagnostic basis for the etiology of a headache. There is level B evidence to suggest that patients requiring an emergent CT are those with a headache and new neurologic findings, those with a new sudden onset of severe headache, and *human immunodeficiency virus* (HIV) positive patients with a new headache. There are level C recommendations that patients over the age of 50 years without neuro findings may be able to have an urgent CT as an outpatient.

Lumbar puncture is considered the gold standard for diagnosing SAH when xanthochromia is measured by spectrophotometry. CT and LP alone each has its limitations. CT often misses small bleeds or those disguised by bone. CT is unable to identify other etiologies. There is reader variability as well as decreased sensitivity in the setting of anemia and over time. LP can miss unruptured aneurysms, arterial dissections, CVT or pituitary apoplexy. Some patients are difficult. One main advantage is the ability to measure the opening pressure. There is level B evidence that LP should be done even when CT is negative.

The data on when to perform a CT before LP is extremely limited by a lack of prospective randomized controlled trials (RCTs) so the recommendations are currently for level C. However, ACEP recommends

CT first when there are signs of increased intracranial pressure (ICP). Finally, ACEP currently recommends with level B data that emergent angiography is not necessary when CT head and CSF analysis are negative. These patients may be discharged home with close follow-up.

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Case Study 39: Lumbar Compression Fracture

CASE HISTORY

An 80-year-old female is presented to the emergency department with low back pain. The pain began suddenly when she bent over to pick up a book from the floor. On physical examination, she has vital signs of temperature 98.6°F, pulse rate 85 beats/minute, respiration rate 16 breaths/minute and blood pressure 140/80 mm Hg. She has point tenderness over the L5 vertebrae on palpation. Straight leg raise is negative bilaterally and neurovascular examination is intact. X-ray of the lumbar spine shows a compression fracture of the L5 vertebrae.

DISCUSSION

The five lumbar vertebrae are very strong and therefore a fracture in one of these vertebrae is suggestive of severe trauma or pathology in the bone. Osteoporosis is a common cause of lumbar compression fractures in postmenopausal women, whereas violent trauma is the most common cause in younger patients.[1] In the absence of severe trauma, malignancy should be ruled out. Although mortality from these fractures is very rare, morbidity may be severe whether it is a complication from pain and bed rest or neurologic deficits.

The normal function of the lumbar spine is to provide stability and support while walking upright. Injuries will affect the lumbar curvature of the spine and subsequently the curvature of the thoracic and cervical spine. Fractures of the lumbar spine may cause significant disability whether it is through pain or the alterations of posture mechanics. Altering posture may cause secondary pain as well as increased risk for falls.

Different types of fractures may occur as a result of trauma. The Denis system of classification may be used to determine whether or not a fracture is stable. Wedge fractures generally result from malignancy or osteoporosis. These fractures are generally symmetric, but 8–14% of them are lateral wedge fractures. Lap belts in motor vehicles accidents (MVAs) may cause flexion and distraction forces and therefore the posterior columns are injured. This most commonly occurs in children who remain neurologically intact. Finally, burst fractures can occur from axial loads to the spine. These are serious fractures that may lead to neurologic deficit.

In nontraumatic cases, the bone density is diminished to a point where minor accidents can cause a trauma. In this case, the fracture is generally

wedge shaped and will increase kyphosis. In cases of malignancy, metastasis is usually the underlying cause. Other cases may include aneurysmal bone cysts, hemangiomas, and spinal infections resulting in osteomyelitis.

The typical presentation involves midline back pain that is axial, nonradiating, aching or stabbing in quality. Signs or symptoms of neurologic injury should be elicited. Referred pain may also be present in ribs, hip, groin or buttocks. Pain is not always present especially in cases involving osteoporosis. Neurological examination should include assessment of rectal tone. Spine curvature should be assessed and vertebrae should be palpated. Site of pain often correlates to the site of fracture. Hip flexor contractures should also be assessed. Differential diagnosis includes coccyx pain, lumbar degenerative disk disease, lumbar facet arthropathy, lumbar spondylolysis and spondylolisthesis, mechanical low back pain and osteoporosis.

WORKUP

Workup should include both blood tests and imaging. Blood tests should include complete blood count (CBC), prostate-specific antigen (PSA) and erythrocyte sedimentation rate (ESR). Urine may be tested for Bence-Jones protein. Initially plain films are the standard. Minimally anteroposterior and lateral views of the lumbar and thoracic spines should be obtained. If possible, lateral flexion and extension views with standing should be obtained to look for gross instability and burst fractures. CT scan may be further utilized to assess the severity of the fractures as well as rule out middle column and burst fractures that are not as easily seen on plain films. CT is also the best method for evaluations of fractures of posterior elements and laminae of the neural arch. When spinal cord injury is suspected magnetic resonance imaging (MRI) must be done. MRI is also helpful for the evaluation of hemorrhage, tumor and infection. Dual energy radiographic absorptiometry (DRA) and positron emission tomography (PET) scanning are also considering, but not likely to be done emergently.

MANAGEMENT

Management may be either operative or nonoperative. Appropriate consults to either orthopedics and neurosurgery, or rheumatology should be made. Nonoperative patients require pain management, bracing and physical therapy. One method of bracing is the thoracic-lumbar-sacral orthosis (TLSO). Physical therapy includes early mobilization and weight-bearing exercises. This usually begins on an inpatient basis and

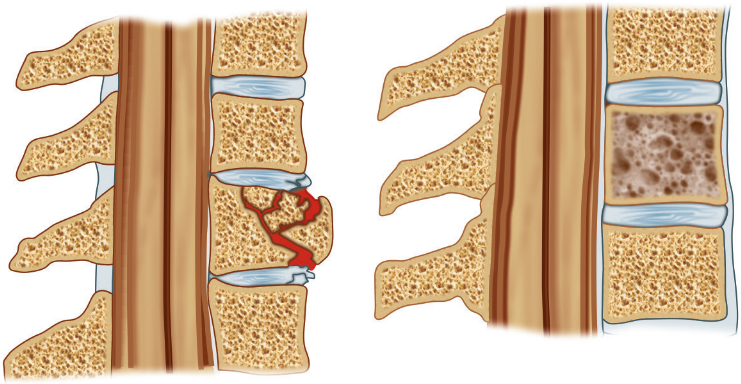

Fig. 1: Transcutaneous vertebroplasty

progresses to outpatient. Monitoring with plain films is required on a monthly basis to screen for fractures that worsen to the point where they require surgical intervention.

Surgery is indicated when there is either neurological dysfunction or instability. Surgical technique depends upon the type of fracture and the overall health of the patient. If a patient continues to experience pain from a wedge fracture after conservation management then a vertebroplasty may be considered (Fig. 1). This procedure involves injecting a cement polymer into the fractured vertebral body. This procedure has proven promising with the possibility of cement going to surrounding structures. A similar procedure called kyphoplasty involves injecting a balloon before cement and, therefore, the cement is injected into a close balloon. Severe fractures will not permit the injection of the balloon. Despite the risks, these procedures have many benefits and are being used more frequently as well as earlier in the treatment process. A brace is generally used postoperatively with the time frame done on a patient-by-patient basis.

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CHAPTER

6

Neurology

Case Study 40: Myasthenia Gravis

CASE HISTORY

History of Present Illness

The patient is a 54-year-old female who is presented to the emergency department with droopy eyelids and requires to lift them in order to see. The patient has weak legs, thus requiring assistance to be mobile. She has difficulty in chewing meat and other hard foods. Family members state that over the years, she slowly became more fatigued, weak, believes that it is a constant low feeling now. She also reports that her symptoms seem to improve after a good night of sleep.

Past Medical History

Before the onset of ptosis, an initial onset of diplopia prompted an eye examination in early 2011. An ophthalmologist found no neurological or muscular abnormality. She was given a few trials of botox injections which did not resolve ptosis and in fact made it worse. Since the patient was not able to pay her medical bills from the prior botox injection, the patient did not seek further medical workup.

Family History

Negative

PHYSICAL EXAMINATION

Neurologic Examination

- *Mental status*: The patient is alert and oriented to time, place and person. Cranial nerves II–XII are intact. When asked to smile, snarling appearance is noted. The patient has mushy speech.

- Sensation and cerebellar function are intact.
- Deep tendon reflexes (DTR) are 2+ and intact.
- Weakness in extremities is noticed when asked to clench fist or when asked to resist pushing anterior region of calf.

LABORATORY TEST RESULTS

- *Complete blood count*: It showed no anemia, leukocytosis, or thrombocytopenia.
- *Basic metabolic panel*: Electrolyte, glucose, blood urea nitrogen (BUN) and creatinine are within normal limits.
- *Urinalysis*: Urine culture showed no hematuria, pyuria and bacteriuria
- *Liver function test*: Aspartate transaminase (AST), alanine transaminase (ALT), bilirubin and alkaline phosphatase are within normal limits.
- Amylase and lipase are within normal limits.
- *Cardiac markers*: Creatine kinase-muscle and brain (CK-MB), troponin and myoglobin are within normal limits.
- *Drugs*: Negative for illicit or over-the-counter (OTC) drugs.

DIAGNOSTIC ADJUNCTS

Computed Tomography

- *Brain*: Rules out intracranial mass, stroke and skull fracture
- *Chest*: Rules out masses

X-Ray

Rules out the thymoma

DIFFERENTIAL DIAGNOSIS

- Senile ptosis
- Lambert-Eaton syndrome (increasing muscle strength on repetitive contraction, Small Cell Carcinoma- Lung)
- Botulism (pupils dilated and repetitive nerve stimulation, incremental increase in muscular fiber contraction)
- Intracranial mass lesion
- Mitochondrial myopathy
    - Oculopharyngeal muscular dystrophy
    - Muscular dystrophies

DIAGNOSIS TESTS

- *Initial test*: Anti-Acetylcholine receptor (Ach-R) antibodies (Ab)

- *Edrophonium (Tensilon)*: Sensitive and not specific
- *Side effects from edrophonium*: Nausea/Diarrhea, fasciculation, bradycardia and syncope
- *Imaging*: X-ray and CT scan
  - *Thymoma*: 10–15%
  - *Thymic hyperplasia*: 65%
- *Accurate test*: Electromyography (EMG) showed decrease in muscular fiber contraction on repetitive nerve stimulation.

IMPRESSION

- *Myasthenia gravis*: Patient presents with muscle weakness and fatigue (Fig. 1).
- Initially she had diplopia and now prominent ptosis and difficulty in swallowing.
- Her speech is mushy with nasal voice.
- Facial weakness with snarling appearance is noted when smiling.
- The disease progressed due to lack of a correct diagnosis and management, leading to generalized weakness involving proximal muscles in an asymmetric pattern.
- Sensation and deep tendon reflexes are intact.
- No evidence of respiratory muscle being affected.
- After intravenous (IV) injection of 30 mg of physostigmine three times in 24 hours, the patient shows a dramatic improvement of ptosis and motor activity.

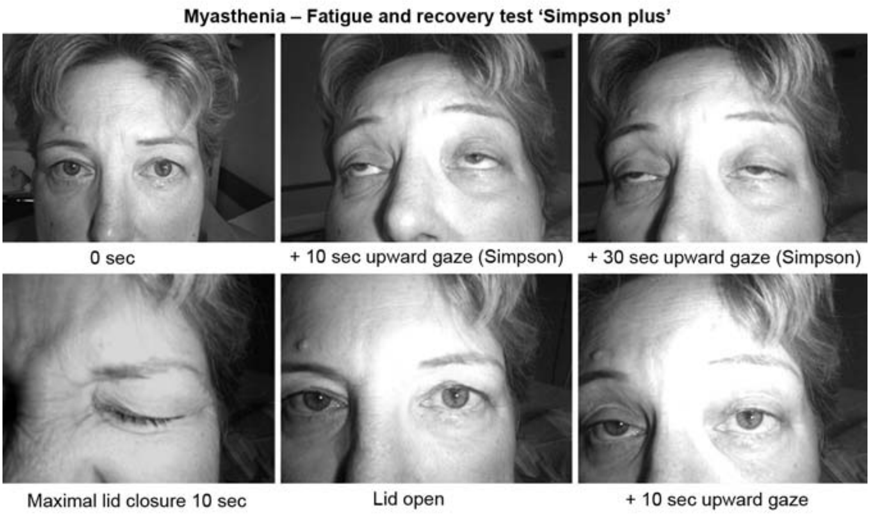

Fig. 1: Myasthenia gravis

Source: Available online from http://www.neurology.org/content/67/8/1524.full

TREATMENT

- *Anticholinesterase drugs:* Pyridostigmine, neostigmine (used for symptomatic treatment).
  - Indicated in thymectomy
  - Used if thymoma present
  - Used before toxic immunosuppressive therapy.
- *Immunosuppressive therapy*: Used if thymectomy not effective.
  - *Glucocorticoids*: Improve weakness, 1–3 months to see effect.
  - Steroids first, but if ineffective, consider:
    - *Combination*: Azathioprine (3–6 months for effect) + Steroid
    - *Cyclosporine and cyclophosphamide*: Alternative to azathioprine; more toxic.
    - *Mycophenolate*: Newer immunosuppressive drug; less side effects than steroids or cyclophosphamide.
    - *Plasmapheresis*: IV immunoglobulin (Ig), rapid improvement of weakness.

ACUTE MYASTHENIC CRISIS

Respiratory involvement

AVOID

Aminoglycoside antibiotics exacerbate MG

FOLLOW-UP

Ptosis and generalized muscle weakness improved with physostigmine injection. Patient is being treated daily with anticholinesterase and sustained-release tablets.

DISCUSSION

Myasthenia gravis is the most common autoimmune disorder of neuromuscular junction (NMJ). Symptoms of ptosis, diplopia, and general weakness are caused by development of autoantibodies to postsynaptic AChR. Symptoms worsen with muscle use. Reversal of symptoms with AChE inhibitors.

Lambert Eaton Syndrome: Autoantibodies to presynaptic Ca^{2+} channel resulting in decreased ACh release. It causes proximal muscle weakness usually associated with paraneoplastic syndromes (small cell lung cancer). The symptoms improve with muscle use and there is no reversal with AChE inhibitors.

PRACTICE PEARL

As compared to normal patients, the dose of succinylcholine for inducing anesthesia in patients with myasthenia gravis should be 2.6 times that of the normal dose.

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Case Study 41: Guillain-Barré Syndrome

CASE HISTORY

Chief complaint: A 14-year-old Caucasian male presents with complaints of bilateral (BL) lower extremity weakness 3 hours prior to admission.

History of Present Illness

When the patient got out of bed early morning, suddenly he felt some weakness and was not able to walk, patient started feeling BL lower leg weakness with tingling at times. There was less feeling on the right, with weakness in both legs. The patient reports no recent trauma, injuries, pain, headache, or other complaints. The patient did not have symptoms previously and has not recently been seen or treated by a doctor or hospitalized Parents state that the patient had four vaccinations 1 week ago.

Past History

Past history is negative.

PHYSICAL EXAMINATION

General Appearance

Alert and anxious 14-year-old white male is not in acute distress and interacts very well with family members and staff. His growth and development are appropriate for his age. Vital signs and physical exam unremarkable except for neuromuscular exam.

Musculoskeletal Examination

The patient's back is symmetrical, no masses found and appears nontender.

Neurologic Examination

- The patient is alert and oriented with good eye contact and pupils are equal, round, reactive to light and accommodation (PERRLA). Babinski's sign is absent.
- Decreased BL lower extremity strength, reflexes and proprioception.
- Cerebellar function is normal.

Extremities/Skin Examination

No joint effusions, tenderness, rashes, edema, or cyanosis found. Capillary refill rate is normal. The patient's skin is warm, dry, intact with no rash and good turgor.

Lab Assessment

LP: Increased protein, with fewer than 10 cells/mm^3.

DIAGNOSTIC ADJUNCT

Computed tomography (CT) of lumbar spine was ordered stat which shows negative findings.

IMPRESSION

Guillain-Barré syndrome: Patient presents with sudden and acute onset of BL lower extremity weakness which disables the patient from walking with decreased reflexes and proprioception. He feels weakness with absence of reflexes, fever and constitutional symptoms (see Fig. 1).

DIFFERENTIAL DIAGNOSIS

- *Basilar artery occlusion:* Asymmetric limb paresis
- *Botulism:* Descending paralysis
- *Transverse myelitis:* Abrupt BL leg weakness and ascending sensory loss
- *Poliomyelitis:* Purely motor neuron destruction of the anterior horn cells
- *Polymyositis*: Chronic, affects proximal limb muscles
- Toxic neuropathy
- Acute Intermittent Porphyria-Abdominal weakness, psychosis and abdominal pain
- Recent diphtheria
- *Vasculitic neuropathies:* In GBS, CSF protein levels elevate without elevating pleocytosis in about 80–90% of patients. CSF in Lyme or HIV disease may have pleocytosis demonstrating analogous meningeal reaction. If white blood cell count in cerebrospinal fluid (CSF) consists of elevated protein higher than 0.55 gm per dL (5.5 gm per L) that increases the possibility of Lyme disease, neoplasia, HIV and sarcoid meningitis.

DIAGNOSTIC TESTS

- *Best initial test:* Lumbar puncture for CSF protein and cell count.
- *More than 48 hours after onset of symptoms:* Elevated protein without rise in cell count.
- *Most accurate test:* Electromyography (EMG) which detects evidence of demyelination of peripheral nerves.

DIAGNOSTIC CRITERIA

AAFP Diagnostic Criteria for Typical Guillain-Barré Syndrome

Features required for diagnosis:

- Progressive weakness in both arms and legs
- Areflexia

Features strongly supporting diagnosis:

- Progression of symptoms over days, up to four weeks
- Relative symmetry of symptoms
- Mild sensory symptoms or signs
- Cranial nerve involvement, especially bilateral weakness of facial muscles
- Recovery beginning two to four weeks after progression ceases
- Autonomic dysfunction
- Absence of fever at onset
- High concentration of protein in cerebrospinal fluid, with fewer than 10 cells per cubic millimeter
- Typical electrodiagnostic features
- Features excluding diagnosis
- Diagnosis of botulism, myasthenia, poliomyelitis, or toxic neuropathy
- Abnormal porphyrin metabolism
- Recent diphtheria
- Purely sensory syndrome, without weakness

TREATMENT

Treatment is given as soon as possible because it becomes ineffective after 2 weeks from the onset of symptoms.

- Intravenous immunoglobulin (Ig) or plasmapheresis is equally effective, no benefit to combination treatment.
- *Glucocorticoids*: Not effective in treatment of acute Guillain-Barré syndrome (GBS).

MANAGEMENT

- Monitor vital capacity in patient with GBS.
- Figure 1 shows the progression of the disease in different stages.
- Initiate early respiratory support to prevent death from respiratory failure.

Guillain-Barré Syndrome

Guillain-Barré syndrome is an acute polyradiculopathy due to an autoimmune destruction of myelin (Fig. 2). There is misdirection of immune response due to molecular mimicry. Patient presents with rapidly developing weakness that begins in the lower extremities and moves upward. Patient lacks reflexes in affected muscle groups. Progression of symptoms develop over the course of hours to days. Legs are usually more affected than arms and face. Fever, constitutional symptoms, or bladder dysfunction are rare. Sensory disturbances can cause pain or tingling dysesthesia. Sensory changes are due to loss of large sensory fibers, resulting in reflex and proprioception loss. Autonomic instability occurs in severe GBS, requiring patient treatment in intensive care unit (ICU). Seventy-five percent of patients with GBS have a history of infection 1–3 weeks prior to onset of symptoms. The infection is typically in the respiratory or gastrointestinal system (Campylobacter jejuni). It may be preceded by infections with human herpes virus, cytomegalovirus and Epstein-Barr virus. Recent formulations of influenza vaccine are associated with one case of GBS per million patients immunized. GBS occurs more frequently in patients with HIV, systemic lupus erythematosus (SLE) and lymphoma.

CSF findings: CSF is often normal when symptoms have been present for less than 48 hours, but by the end of the first week, CSF protein level is greater than 5.5 g/L with no pleocytosis (less than 10 cells/cm).

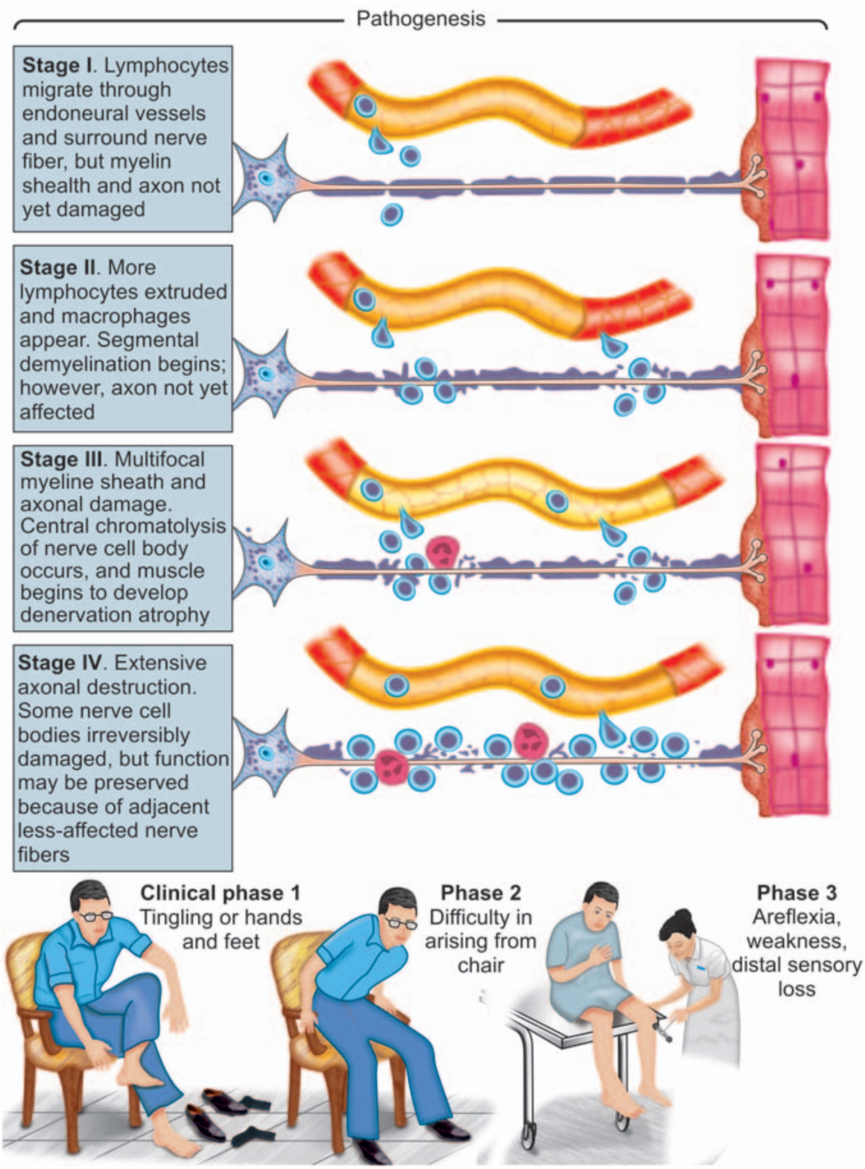

Fig. 1: Clinical stages/Phases of GBS

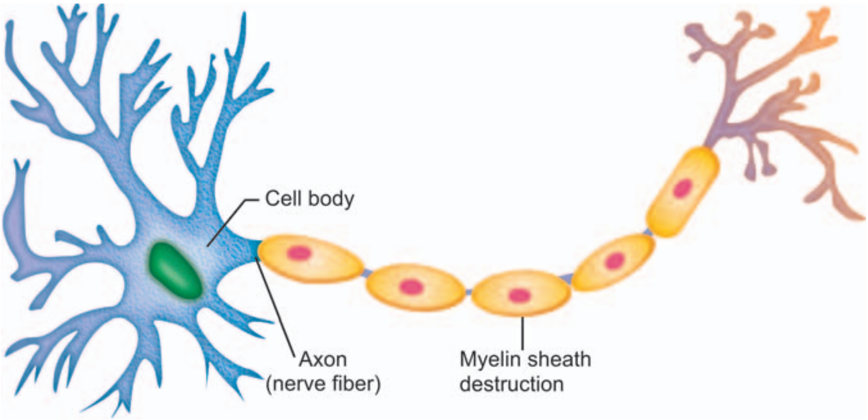

Fig. 2: Destruction of myelin sheath in Guillain-Barré syndrome

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Case Study 42: Delirium Tremens

CASE HISTORY

A 67-year-old gentleman was arrested on a Saturday night for drunk driving, received his seventh driving under the influence (DUI) violation, and was driving with a revoked license. He was taken to the local jail. The following afternoon (Sunday), he began to pace around his jail cell and became agitated and tremulous. Then, on Monday afternoon, he became increasingly anxious and had several episodes of vomiting. Tuesday morning, he did not eat due to additional nausea and vomiting. In the early afternoon on Tuesday, he had a generalized seizure of approximately 45 seconds and was taken to the emergency room (ER). On the way to the hospital, he remained mildly confused about the situation, but did not have any signs of head trauma. In the ER, he was initially found to be quite anxious with mild tachycardia and hypertension.

EMERGENCY ROOM MANAGEMENT

Intravenous (IV) fluids like normal saline (NS) were started immediately and he was given thiamine, naloxone and glucose. Over the following hours while he was being assessed, he became progressively more agitated and confused about where he was, began having hallucinations of people telling him that he must get out of town, and he developed severely worsening tachycardia and hypertension.

He was given a bolus of diazepam and was transferred to the detox unit. There, he was monitored with the revised clinical institute withdrawal assessment for alcohol (CIWA-Ar) protocol using Ativan (lorazepam) for the following 4 days as he progressively returned to baseline and required decreasing amounts of Ativan. Upon discharge, he agreed to attend an alcoholic rehabilitation program.

BRIEF CASE DISCUSSION

This patient is a chronic and heavy alcohol user, which is evidenced by his multiple DUIs with revoked driver's license and his progression to withdrawal symptoms. Although not usually the case, this patient demonstrated a progression through the various forms of alcohol withdrawal: minor withdrawal, major withdrawal, withdrawal seizure, and delirium tremens (DTs). Chronic alcohol use affects both the gamma-aminobutyric acid (GABA) and N-methyl-D-aspartate (NMDA) receptors in the brain, with counter-effects occurring when alcohol is removed. This

combination leads to the symptoms associated with alcohol withdrawal. Treatment is generally aimed at preventing adverse effects and additional withdrawal episodes. Progression to DTs can have a high morbidity and mortality, so it is important to recognize and treat quickly, while monitoring the patient through the withdrawal and detoxification stages. Finally, preventing future recurrences should be addressed by assessing willingness to change and providing information regarding rehabilitation opportunities.

DELIRIUM TREMENS DISCUSSION

General

Alcohol withdrawal syndrome can manifest with variable symptoms and occurs as a result of abrupt alcohol cessation in an alcohol-dependent individual. These include minor withdrawal, major withdrawal, withdrawal seizures and DTs. These do not have to occur in any particular order, nor do they all even have to present at all. Minor withdrawal generally occurs within the 1st day after alcohol cessation and includes symptoms, such as tremor, anxiety, nausea, vomiting and insomnia. Major withdrawal usually presents within 2–3 days of the last drink, exhibited by potential visual and auditory hallucinations, whole body tremor, vomiting, diaphoresis and hypertension. Withdrawal seizures also may occur within the first 2–3 days. These tend to be brief, generalized, motor seizures in those who do not have a history of seizures. In some cases, this can be the initial and only symptoms during alcohol withdrawal. Without treatment, as many as 60% can have multiple seizures, but this rarely develops into status epilepticus. As many as 30–40% of people experiencing withdrawal seizures may progress to DTs. This is the most severe form of alcohol withdrawal, which tends to occur within the first week after alcohol cessation and can include symptoms such as agitation, global confusion, disorientation, hallucinations, fever, hypertension, diaphoresis and autonomic hyperactivity. Risk factors for developing DTs include prior withdrawal seizures or DTs, concurrent illness, prolonged heavy daily alcohol consumption, current severe withdrawal symptoms, or prior detoxification. While less than 50% of alcohol-dependent people experience severe withdrawal needing pharmacological intervention, only about 3% will progress to DTs.

Pathophysiology

Alcohol has effects on several receptors within the central nervous system. These include both the inhibitory GABA receptor and the excitatory NMDA receptor. Alcohol activates the GABA receptor and inhibits the

NMDA receptor. With chronic alcohol use, this leads to downregulation of the GABA receptor and upregulation of the NMDA receptor. Upon removal of the alcohol stimulus, there will be less inhibitory GABA stimulation and more excitatory NMDA stimulation, which both lead to an increase in neuroexcitation. The combination of these effects leads to the mental confusion and autonomic hyperactivity associated with the various stages of alcohol withdrawal syndrome. The longer a person is exposed to chronic alcohol, more alterations occur with the GABA and NMDA receptors. This leads to increased frequency and severity of withdrawal episodes over time, which is a phenomenon known as kindling.

Assessment

It is important to obtain a detailed history of the patient's situation. Remember that everything should not be blindly attributed to alcohol since people with alcohol dependence can also have other illness. With this in mind, be sure to determine the length of time and amount of regular alcohol intake, the time since the last drink, prior withdrawal episodes or detox admissions, past seizure history, additional medication or substance use or abuse, and any concurrent medical or psychiatric history. A detailed history alone can give you a very good idea of whether the symptoms are related to alcohol withdrawal syndrome. The physical examination is nondiagnostic, so it should be used in conjunction with additional history and laboratory studies to obtain information regarding conditions that may complicate or exacerbate alcohol withdrawal and to ensure that alcohol withdrawal is the most likely cause of the problems. Such physical examination and history information should relate to vital signs (tachycardia, tachypnea, fever and hypertension), general status (tremors, diaphoresis and trauma), the cardiovascular system [arrhythmia, congestive heart failure (CHF) and coronary artery disease (CAD)], the gastrointestinal (GI) system (GI bleeds, liver disease and related stigmata, and pancreatitis), the neurologic system (nystagmus, gait, neuropathy, focal defects, global confusion and altered mental status) and psychiatric issues (disorientation, anxiety, depression, mania and memory). Although none of this is diagnostic for alcohol withdrawal or DTs, it can provide additional insight into other causes or potential complicating features since many people with alcohol dependence have comorbid conditions. Next, laboratory testing and imaging should be used similarly to investigate complicating factors and to optimize the potential for swift recovery. Serum blood alcohol can be an important value to obtain, since withdrawal symptoms with alcohol still in the body would suggest that the withdrawal will worsen as the alcohol continues to decrease. Liver and renal function tests should be obtained since this

can potentially complicate or exacerbate the withdrawal. Furthermore, a drug screen can be utilized if there is suspicion of additional drug use. A complete blood count with differential and comprehensive metabolic panel should be obtained to evaluate potential concurrent infection, electrolytes and blood glucose. Electrolytes and glucose should be corrected to minimize complications. Additionally, if concurrent infection is suspected, then investigation into the location should occur. Infection can greatly increase morbidity during withdrawal and DTs. Potential studies may include chest radiograph, urinalysis, lumbar puncture, and more as deemed appropriate. Finally, computed tomography (CT) of head or cervical radiography may be utilized if head or neck trauma is evident or suspected.

Treatment

Mortality for patients experiencing DTs can be as high as 15%, so it is important to evaluate and treat the patient appropriately in order to minimize complications and facilitate recovery. As previously mentioned, the workup should attempt to rule out any other causes or complicating factors and then treat those accordingly. This includes treating concurrent infections, correcting electrolyte abnormalities and removing any exacerbating medications. Furthermore, many alcohol-dependent patients are also nutritionally deficient and dehydrated. This can be exhibited by inadequate levels of various vitamins, as well as hypoglycemia and ketoacidosis from depleted glycogen stores. Thiamine is a common deficiency in this population and should be replaced along with multivitamins, glucose and IV fluids. Note that it is critical to administer the thiamine prior to giving glucose since glucose utilization can cause a decrease in thiamine and potentially precipitate Wernicke's encephalopathy. The mainstay of assessment and therapy for alcohol withdrawal is the revised CIWA-Ar.[1] This is the most well-validated tool for alcohol withdrawal when used properly in the correct circumstances. First, it is important to be sure that the patient is truly suffering from alcohol withdrawal and not some other cause for decreased arousal, altered mental status, or delirium. Additionally, it has not been validated for complex medical or surgical patients, but instead it should be used for patients in detoxification units, psychiatric units, or medical/surgical wards. CIWA-Ar is a survey or checklist that evaluates 10 items: nausea and vomiting, anxiety, tremor, sweating, auditory disturbances, visual disturbances, tactile disturbances, headache, agitation and clouding of sensorium. Each of the first nine categories is rated on a scale from 0 to 7, while the last item is rated from 0 to 4. With a total (i.e. worst) possible score of 67, any score greater than 15 implies moderate-to-severe

alcohol withdrawal and requires hospital admission and treatment. Mild withdrawal includes scores from 8 to 15, in which pharmacological therapy is still pertinent, but hospital admission may or may not be needed depending on prior history or episodes of alcohol withdrawal. Those with scores under 8 will not need hospital admission or drug therapy for the alcohol withdrawal episode. The utilization of the CIWA-Ar criteria not only assesses the severity of the alcohol withdrawal state, it also guides the treatment. Benzodiazepines, such as diazepam and lorazepam, are given at varying doses and frequencies depending on the severity of withdrawal determined by the CIWA-Ar protocol. Reassessment is continued as the withdrawal state resolves over the course of hours to days. It should be apparent that the patient will begin to require lower and less frequent medication doses as the withdrawal episode improves. It is suggested that the patient is well enough for discharge when three sequential CIWA-Ar assessments deem it no longer necessary for benzodiazepine treatments. Throughout this process, the patient must be monitored closely as complications of alcohol withdrawal and DTs include over sedation, although the most common conditions leading to morbidity and mortality in this group are cardiac arrhythmias and respiratory failure even in the setting of appropriate medical treatment. Patients at highest risk for complications include those with severe fever, fluid and electrolyte abnormalities, concurrent illness, trauma, pneumonia, hepatitis, pancreatitis, alcoholic ketoacidosis, or Wernicke-Korsakoff syndrome. This is an opportunity to assess willingness to change, suggest alcohol rehabilitation opportunities, and introduce possible adjunctive medical therapies (e.g. disulfiram, naltrexone, acamprosate and topiramate). The best possible outcome after such alcohol withdrawal episodes would be supportive in overcoming alcohol-dependence and avoiding any similar episodes in the future.

EXCITED DELIRIUM

The nations emergency rooms are seeing increasing numbers of patients who are using synthetic, designer, drugs. Excited delirium is now more in the news media and sometimes blamed with the use of physical force used by law enforcement agencies, including police restraint and tasers. It is also called Excited Delirium Syndrome, which has a very high rate of mortality, resulting in sudden death from cardiac or respiratory arrest. Head trauma and alcohol withdrawal may be contributing factors to this disorder. But the most common reason is the use of cocaine and bath salts. Bath salts is the informal “street name” for a family of designer drugs often containing substituted cathinones, which have effects similar to amphetamine and cocaine. Their white crystals often resemble

Fig. 1: Bath salts

legal bathing products like epsom salts, but are chemically disparate from actual bath salts. Bath salts packaging often states "not for human consumption" in an attempt to avoid the prohibition of these products.

Other "street names" for this drug are Ivory Wave, Purple Wave, Vanilla Sky, and Bliss. ACEP accepts Excited Delirium as a unique syndrome. The signs and symptoms of excited delirium include severe disorientation, hyperaggression, tachycardia, paranoia, hallucinations, forceful and incoherent speech, severe hyperthermia, and profuse sweating even in cold weather. Most of the time, the person is found naked with all or some of the symptoms described above. Majority of the fatal case reports are of African American males who are involved in acute drug use, especially psychostimulants such as cocaine, PCP, and methamphetamine.

REFERENCE

1. Sullivan JT, Sykora K, Schneiderman J, et al. Assessment of alcohol withdrawal: the revised clinical institute withdrawal assessment for alcohol scale (CIWA-Ar). Br J Addict. 1989;84(11):1353-7.

ADDITIONAL READING

1. Medscape and 5Minute Consult
2. http://en.wikipedia.org/wiki/Excited_delirium
3. http://en.wikipedia.org/wiki/Bath_salts_(drug)

Case Study 43: Stroke

Time lost is brain lost.—American Stroke Association
Time is muscle; time is kidney; time is brain; time is everything in case of emergency

—Badar M Zaheer

CASE HISTORY

A 60-year-old white female is brought to the emergency department (ED) by her husband with sudden onset of right upper extremity weakness that began while she was preparing breakfast in the morning. The husband became concerned when the patient could not talk in response to questions. Screening tools were used and patient seems to understand what is being said but cannot respond (Figs 3 and 4). Patient has history of medication-controlled hypertension. The patient's father died of a stroke at the age of 70 years after living his whole life with hypertension, but her mother lived to the age of 80 years completely healthy.

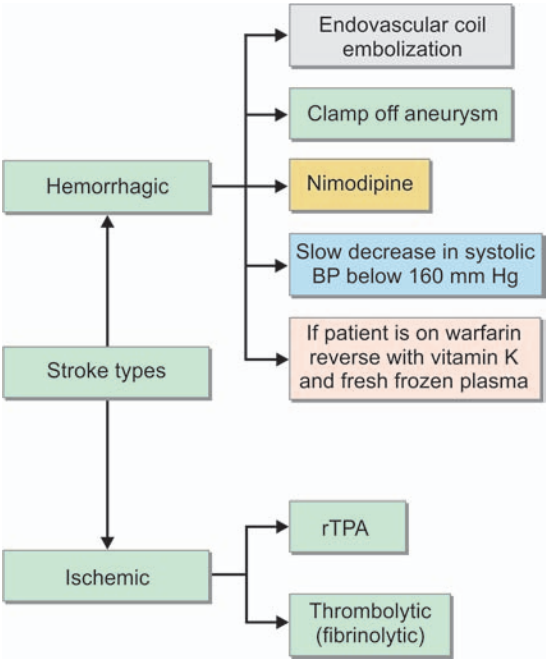
PHYSICAL EXAMINATION

- *Neurologic examination*: Cranial nerves (CN) II-VI, VIII-XII are intact with right facial nerve palsy. She can wrinkle her forehead. Droop noted on the right side of the mouth. No lid weakness noticed.
  - *Motor*: Flaccid hemiparesis noted onto the right side.
  - *Sensory*: No light touch of right upper extremities (RUE).
  - *Reflexes*: No deep tendon reflexes (DTR) of right lower extremities (RLE). Babinski's reflex is present on the right big toe. Corneal and gag reflexes are normal.
  - *Cerebellar*: Slight truncal ataxia to the right side and gait disturbance.
  - *Visual/Neglect*: Lost vision and neglect on the right side of the eye.
  - *Language*: Dysarthria, expressive aphasia and no receptive aphasia.
  - *Level of consciousness*: Slightly somnolent and responds to verbal stimuli.
  - Rest of the physical examination is normal.

DIAGNOSTIC WORKUP

- Initial (most sensitive) test available to detect blood in the brain: Noncontrast head computed tomography (CT) scan. See Flow chart 1 for hemorrhagic versus ischemic stroke.
  - Negative for ischemia within first 48 hours after onset of symptoms.
- *Accurate test for ischemia:* Available
- Diffusion-weighted magnetic resonance imaging (MRI) is done for cerebral ischemia detection.

Flow chart 1: Types of stroke



- Search for embolic source by doing an echocardiogram, carotid duplex scan and 24-hour Holter monitoring.
- Initial blood test is done to check for inherited hypercoagulability.
- Electrocardiogram (ECG) may help to show ischemia or inverted T-waves.
- Bubble study on echocardiogram to detect cardiac defect and function. The bubble echocardiogram shows better results than a conventional echocardiogram. Watch on Youtube http://www.youtube.com/watch?v=3dssbDeow50

MANAGEMENT

A diagnosis of acute ischemic stroke and acute onset of focal neurologic deficit is made. The course of treatment suggested for this patient was tissue plasminogen activator (tPA) since there were no hemorrhages found and it was within 2 hours of the onset of symptoms. The patient was administered tPA: 0.9 mg/kg body weight. Follow the guide-lines from: http://www2.massgeneral.org/stopstroke/tpaDoseCalc.aspx. A repeat neurologic examination was performed 90 minutes following treatment and showed increased speech and use of the right arm. The patient was

discharged to a rehabilitation hospital on Day 7 and scheduled for a follow-up examination in 3 months.

Acute Ischemic Stroke

- Acute onset from a focal neurological deficit
- *CT*: No low density areas of bleeding
- No contraindications (CI) to tPA, blood pressure (BP) is less than 185/110 mm Hg.
- No family to defer tPA use. tPA is administered, with no complication.
  - Consent for tPA is necessary.
  - Explain and elaborate on the risks and benefits of tPA use.
  - Received verbal consent and signature with left hand.
  - tPA administered in less than 2 hours after cerebrovascular accident (CVA) symptoms onset.
  - *Initial bolus*: 5 mg slow IVP over 2 minutes.
  - *Follow-up infusion*: 40 mg infusion over 60 minutes.
  - Repeat neurological examination at 90 minutes.
  - *Repeat examination*: Increased speech and use of right arm.
  - Decreased mouth droop and visual neglect.
- *Hospital course*: No hemorrhage, and improved neurological function.
  - *Disposition*: Rehabilitation in hospital on Day 7. Follow-up examination in 3 months. Near complete use of RUE. Speech and vision improved. Slight residual gait deficit. Her husband is at home to assist.
  - POEMs: Patient Oriented Evidence that Matters.

Question: Does adding endovascular procedures to intravenous tissue plasminogen activator (t-PA) improve outcomes for patients with stroke?

Answer: Local delivery of t-PA extracting the thrombus or stenting (endovascular therapies) showed no benefit in a randomized clinical study and in other two clinical trials which reaffirmed the ineffectiveness of this procedures (Figs 1 and 2). (N Engl J Med. 2013;368(10):904-914) (Kidwell CS, Jahan R, Gornbein J, et al. A Trial of Imaging Selection and Endovascular Treatment for Ischemic Stroke. N Engl J Med. 2013; 368(10):914-923).

None of the above procedures are as effective as intravenous t-PA which provides modest benefit, if the patients are rightly selected.

DIFFERENTIAL DIAGNOSIS

It includes epileptic seizures and postictal subdural hematoma, tumor, hyponatremia, hypocalcemia, hepatic encephalopathy, Wernicke Korsakoff syndrome, hypoglycemia, hyperglycemia, alcohol, illicit drugs, head

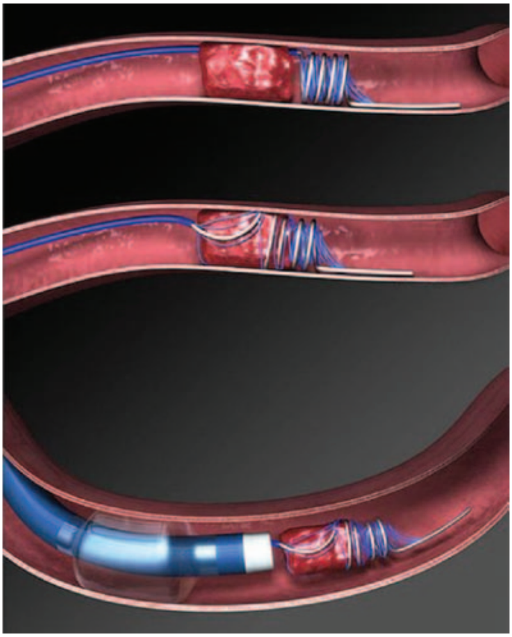


Fig. 1: Mechanical thrombectomy
Courtesy of Wikipedia: http://en.wikipedia.org/wiki/File:Merci_L5.jpg

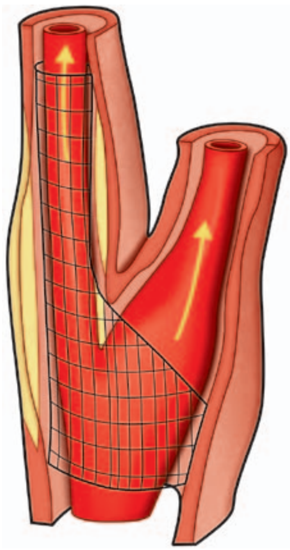


Fig. 2: Stents and angioplasty
Courtesy of Wikipedia: http://en.wikipedia.org/wiki/File:Merci_L5.jpg

injury, encephalitis, cerebral abscess, hypertensive encephalopathy, peripheral nerve lesion, multiple sclerosis, Creutzfeldt-Jakob disease, and subarachnoid hemorrhage.

TREATMENT

Golden Hour of Acute Ischemic Stroke (Fig. 5)

Acute ischemic stroke is an extremely serious medical emergency. In a typical large-vessel acute ischemic stroke, 1.9 million neurons may be lost each minute without medical management. Rapid intervention is crucial in the management of acute ischemic stroke. Door to treatment in less than or equal to 60 minutes is the standard of care recognized by professional medical associations involved in the treatment of acute ischemic stroke (Fig. 5).

- *Tissue plasminogen activator*: Administered in less than 3 hours from onset of symptoms, better neurologic function in 3 months post-CVA.
- *Contraindication to tPA*: Stroke or serious head trauma less than 3 months ago.
  - *Hemorrhage:* Gastrointestinal/genitourinary (GI/GU) less than 3 weeks ago.
  - Surgery less than 2 weeks ago
  - History of intracranial hemorrhage
  - Arterial puncture less than 1 week ago
  - Lumbar puncture less than 1 week ago
  - Blood pressure more than 185/110 mm Hg

Fig. 3: Screening Tool; Patients must be able to identify the problems in the situation
Source: www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.

Fig. 4: Screening Tool; Patients are asked to identify the objects in the picture
Source: www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.

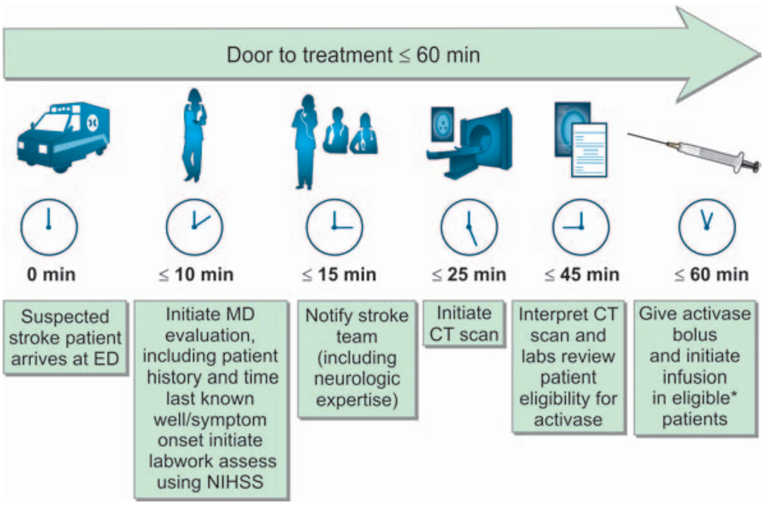


Fig. 5: Golden Hour for Thombolytic Therapy
Source: Adapted from http://www.ninds.nih.gov/news_and_events/proceedings/stroke_proceedings/recs-emerg.htm#emergency; and Jauch EC. American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2010;122(18 suppl 3):S818-S828.

Checklist before Starting tPA Therapy

1. Check the indications below that apply:
   - ☐ Patient is 18 years of age or older
   - ☐ Time of symptoms onset can be identified accurately
   - ☐ Thrombolytic therapy can be started within 3 hours of symptom onset.
2. If all the boxes in Step 1 are checked, then review the absolute contraindications below and check the ones that apply:
   - ☐ Head CT scan today showing intracranial bleeding
   - ☐ Head CT scan today shows no intracranial bleeding but the clinical presentation is suspicious for subarachnoid hemorrhage
   - ☐ Head CT scan today shows multilobar infarction (hypodense area greater than one-third the area of the cerebral hemisphere)
   - ☐ Any of the following within the past 3 months; intracranial or intraspinal surgery, serious head trauma, or a witnessed seizure
   - ☐ Witnessed seizure since the onset of symptoms
   - ☐ Blood pressure higher than 185 mm Hg (systolic) or higher than 110 mm Hg (diastolic)
   - ☐ Arterial puncture at noncompressible site within past 7 days.

   Risk of hemorrhage:
   - ☐ Evidence of active internal bleeding
   - ☐ Patient has an arteriovenous malformation, aneurysm, or neoplasm
   - ☐ Prior history of intracranial bleeding
   - ☐ Laboratory evidence of a coagulopathy (e.g. platelet count < 100,000/μL)
   - ☐ Patient on coumadin and INR greater than or equal to 1.7, or patient received heparin in past 48 hours and aPTT above normal range.
3. If none of the boxes in Step 2 are checked, review the relative contraindications below and check any that are considered an unacceptable risk:
   - ☐ Major surgery or serious trauma in past 14 days
   - ☐ Gastrointestinal or urinary tract bleeding within past 21 days
   - ☐ Acute MI in past 3 months or post-MI pericarditis
   - ☐ Blood glucose less than 50 mg/dL or more than 400 mg/dL.

If all boxes in Step 1 are checked, and no boxes in Step 2 and 3 are checked, then give thrombolytic therapy.

Source: Adapted from American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 9: Adult Stroke. Circulation. 2005;112:IV111-20.

- *Current use of anticoagulants*: Platelets less than 100,000/mm^3 coagulopathy, prothrombin time (PT) is more than 15 seconds.
  - *Heparin*: It is given but no clear benefit
  - Increased risk of bleeding.
  - *Strongest indication*: Acute thrombosis
  - *Used only with higher risk of recurrent stroke*: Risk from side effects offsets benefit of treatment.
- Atrial fibrillation, basilar artery thrombosis, stroke in evolution

Antiplatelet Therapy

- *Aspirin*: It is a first line drug most useful in secondary prevention of ischemic stroke.
- 24 hours post-tPA, dipyridamole, if continue to have recurrent CVA on aspirin alone
- *Clopidogrel*: Known allergy to aspirin

Surgical Interventions

- *Carotid endarterectomy*: Occlusion of less than 70% of arterial lumen and symptomatic lesion. Mechanical Thrombectomy (see Fig. 1).
- *Alternative*: Carotid stenting (see Fig. 2).

Practice Pearl

The Emergency MD needs to be able to diagnose accurately and acts upon certain CT findings without specialist (e.g. radiologist) assistance, because many disease processes are time-dependent and require immediate life-saving action. Don't forget the mnemonic while reading the CT

B	Blood	Blood
C	Can	Cisterns
B	Be	Brain
V	Very	Ventricles
B	Bad	Bone

head—"Blood Can Be Very Bad," where blood = blood, can = cisterns, be = brain, very = ventricles, and bad = bone.

Blood

- Presence of blood, its location, and spread (Figs 6 to 8).
- Acute bleeding absorbs X-rays and they become white (Hyperdense).

Cisterns

Examine the cisterns which are collections of CSF protecting the brain. Look for asymmetry, presence of blood, and effacement.

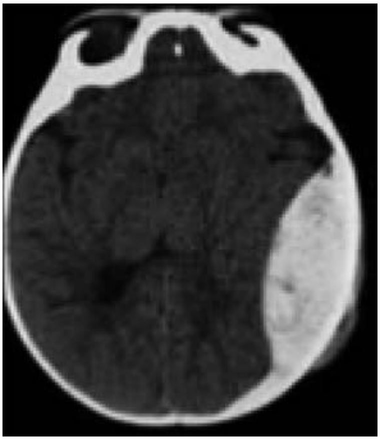


Fig. 6: Epidural hematoma: Elliptical/lens shaped

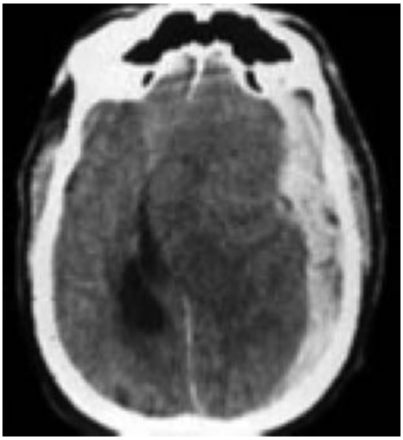


Fig. 7: Subdural hematoma: Crescent

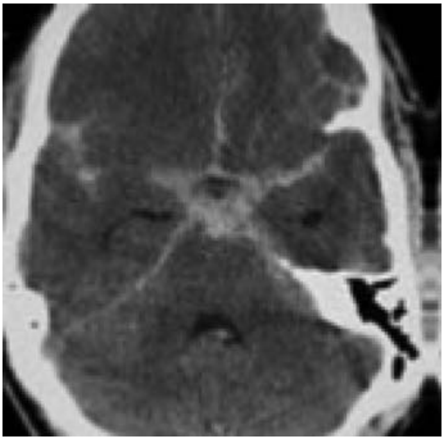


Fig. 8: Subarachnoid hemorrhage: Star shaped, Texas "The Lone Star Flag"

Fig. 9: Warning Signs of Stroke National Stroke Association

Brain Matter

- Look for asymmetry and midline shift. Trace the falx as your guidance.
- Identify white hyperdense areas associated with blood IV contrast, or any calcifications.
- Look for hypodense areas which are associated with fat, air, ischemia or tumor.

Ventricles

Look at third and fourth ventricles for asymmetry and dilatation and for hemorrhage.

Bone

On CT scans, cortical bones of the skull have the highest density. It is best viewed on separate bony windows when looking for fractures or tumors.

Public Awareness and Public Education

The accronym FAST should be used to educate the public about stroke (Fig. 9).

ADDITIONAL READING

1. NIH Stroke Scale http://nihss-english.trainingcampus.net/uas/modules/trees/windex.aspx.
2. http://lifeinthefastlane.com/education/investigations-tests/ct-head-scan/ American Stroke Association.
3. http://www.activase.com/iscstroke/golden.

Case Study 44: Bell's Palsy

CASE HISTORY

A 30-year-old male is presented to the emergency department (ED) with a sudden onset of right-side facial weakness and right eye irritation. He woke up with these symptoms. He denies any preceding history of viral syndrome or trauma. Past medical history is negative.

Diagnosis

Facial nerve palsy (Bell's Palsy) (Fig. 1).

SIGNS AND SYMPTOMS

Common signs are excessive tearing, unilateral facial drooping and inability to wrinkle the forehead.

Discussion

Bell's palsy is a diagnosis of exclusion. Other causes of facial nerve palsy include Lyme disease, tumors of the temporal bone, Ramsey Hunt syndrome and acoustic neuromas. The onset of Bell's palsy is sudden, and the symptoms can range from weakness to complete paralysis for more than a week; however, sensations are intact. It is usually preceded by a viral infection but not always. Associated symptoms may include pain behind the ear, ipsilateral loss of taste sensation, decreased or increased lacrimation and hyperacusis. Majority of the patients recover within a few months. However, if the symptoms do not resolve in 6 months, laboratory studies, imaging studies or motor-nerve conduction studies should be considered. Bell's Palsy is secondary to complete destruction of the facial nerve nucleus (Lower Motor Neuron lesion) itself or its facial nerves' branches. Whereas upper motor neurons of the motor cortex or connection between cortex and facial nucleus show contralateral paralysis of lower face only.

Treatment

- Use an eye patch while sleeping to prevent corneal drying and abrasions.
- Frequent application of artificial tears while awake.
- Massaging the weakened muscles might improve muscle tone.
- Corticosteroids and antiviral agents such as acyclovir (oral) should be started as soon as possible or at least within a week of the onset of the symptoms.

Fig. 1: Male patient with Bell's palsy
Courtesy: Wikimedia.org.

- If medical therapy is unsuccessful, surgical decompression of the facial nerve might be helpful.

ADDITIONAL READING

1. Signs and Symptoms of Lyme Disease. Centers for Disease Control and Prevention. Available at http://www.cdc.gov/lyme/signs_symptoms/Accessed April 8th, 2013.
2. "Bell's Palsy Fact Sheet," NINDS. Publication date April 2003. All NINDS-prepared information is in the public domain and may be freely copied. Credit to the NINDS or the NIH is appreciated.

Case Study 45: Seizure Disorders in Children

CASE HISTORY

A 5-year-old child is presented in the emergency room (ER) with a chief complaint of seizures which were tonic clonic, lasted less than 5 minutes, and were bilateral. On examination, vital signs are temperature of 104°F, heart rate 120 beats/minute, blood pressure 80/30 mm Hg and oxygen saturation 99% on room air. This is the first episode and the parents were terrified.

Family History

Father had similar episode when he was a child.

DIAGNOSTIC WORKUP

- Septic work-up is done.;
- Blood, urine culture and complete blood count (CBC) were negative.

TREATMENT

Conservative treatment is followed. Ice packs and tylenol are given to control fever and diazepam suppository is also given. The child is more than 18 months, so lumbar puncture is not advisable. No computed tomography (CT) is required. Anticonvulsive drugs are not indicated for simple seizure. Electroencephalogram (EEG) is advised on an outpatient basis.

DISCUSSION

Differential Diagnosis of Seizures in Children

- Fever/Febrile Seizures
- Epidural and subdural infections
- Meningitis or encephalitis
- Epidural hematoma
- Sepsis or bacteremia
- *Epilepsy type*: Rolandic is the most common in children. The diagnosis can be confirmed when the characteristic centrotemporal spikes are seen on electroencephalography (EEG). Typically, high-voltage spikes followed by slow waves are seen. The prognosis for rolandic seizures is invariably excellent, with probably less than 2% risk of developing

absence seizures. Given the benign nature of the condition and the low seizure frequency, treatment is often unnecessary.

Seizures in children are commonly seen in emergency department. Definition of status epilepticus has changed. Previously, Before, we used to define status epilepticus as continuous seizures lasting more than 30 minutes or more than two sequential seizures without full recovery of consciousness in between the seizures. Now, we agree that a shorter period of seizure activity can also result in neuronal injury and that seizure activity is unlikely to terminate by itself after 5 minutes. A duration of longer than 5 minutes is the criteria for status epilepticus especially if the seizure type is generalized convulsive disorder which does not resolve spontaneously in 3–5 minutes. Our major goal in the emergency room is to provide rapid control of the seizure activity in order to prevent neurologic injury. In prehospital, status epilepticus intramuscular (IM) midazolam is equally effective as compared to intravenous (IV) administration of benzodiazepines.

In conclusion, for subjects in status epilepticus, IM midazolam is as safe and effective as IV lorazepam for the hospital seizure physician.

Courtesy: National Institute of Neurological Disorders and Stroke and others and Clinical Trials.gov.

Intravenous Keppra® Levetiracetam

Keppra® can also be effective for acute status epilepticus treatment which is frequently given by neurologists in acute seizure management. However, benzodiazepines are still used as the first line therapy. Intravenous valproic acid (VPA) can be used effectively also for acute seizure control, safely in children. Intravenous VPA is safe and effective in treating acute seizures in emergency department for children. The dose of midazolam is for IM use is 0.2 mg/kg of body weight. Atomized or buccal midazolam can be given in a dose of 0.2–0.5 mg/kg of body weight. When there is a great risk of permanent neurologic injury without delay, it is better to use intraosseous line placement after giving IM, nasal or buccal medication (Versed) when IV line is not possible.

Further Reading

Background: Early termination of prolonged seizures with intravenous administration of benzodiazepines improves outcomes. For faster and more reliable administration, paramedics increasingly use an intramuscular route.

Question: If intravenous access is not available, how good are intramuscular benzodiazepines?

Answer: Intramuscular midazolam was shown in this trial to be an acceptable alternative route for acute seizure control.

ADDITIONAL READING

1. http://www.epmonthly.com/cme/current-issue/pediatric-seizures-/1/.
2. Silbergleit R, Durkalski V, Lowenstein D, et al. Intramuscular versus intravenous therapy for prehospital status epilepticus. N Engl J Med. 2012;366(7):591-600.
3. http://en.wikipedia.org/wiki/Rolandic_epilepsy.

Case Study 46: Seizure Disorders in Adults

CASE HISTORY

A 48-year-old male patient was brought to the ED after being involved in a motor vehicle accident. His vehicle rolled over multiple times and landed in a ditch; the airbag did not deploy. The paramedics noted severe damage to the front of the car. VS: BP = 110/85 mm Hg, HR = 140 beats per minute, R = 24 breaths per minute, saturating 98% on 15 L via nonrebreather mask. During transport to the ED, the paramedics noted a seizure involving tonic clonic movements of all four extremities. Lorazepam 2 mg IV was given and resolved the seizure. Upon arrival, the Glasgow Coma scale score was 8 and blood sugar was 75 mg/dL. Pupils were 3 mm and reactive. Normal tone was present in all four extremities and reflexes were 2 + throughout. The remainder of the exam was unremarkable. A CT head was performed and revealed a large right frontal intraparenchymal hemorrhage. The Emergency MD then spoke to the neurosurgeon and the ER was prepared for the arrival of the patient.

DISCUSSION

Traumatic brain injury results from direct or indirect forces to the brain. Direct injury is caused by the force of an object striking the head or penetrating injury. Indirect injuries result from acceleration and deceleration forces that results in the movement of the brain within the skull.

MANAGEMENT

- Initially stabilize the patient by following ATLS guidelines and administer 100% oxygen.
- Slow and sustained careful blood pressure monitoring is necessary It should be maintained around a mean arterial pressure (MAP) more than 90 mm Hg. If hypertensive, 25% to 30% reduction of MAP may be achieved carefully.
- As intracranial bleed is identified an immediate neurosurgery consult is obtained and the trauma team is alerted and waiting the patient's arrival in the receiving hospital.
- For signs of increased ICP, elevate the head of the bed to 15 to 30 degrees. Maintain MAP greater than 90 mm Hg and maintain adequate arterial oxygenation. If signs of neurological dysfunction, may give mannitol 0.25 to 1 g/kg IV bolus. Hyperventilation no longer

standard of care because it may cause cerebral ischemia. Use only in the short-term as a last resort. Other first line treatments include sedation, CSF drainage, osmotic diuretics. If initial attempts to decrease ICP fail, patient may require emergency decompression by trephination which is rarely done these days.
- For seizure prophylaxis, use anticonvulsants in consult with neurosurgery. May use benzodiazepines, or fosphenytoin with a loading dose of 18–20 mg/kg.
- Use prophylactic antibiotics such as ceftriaxone 1 g IV q 12 hours if patient has basilar skull fracture or penetrating injury.

ADDITIONAL READING

1. Ma OJ. "Head Injury." Emergency Medicine Manual. 6th edition pp. 774-9.

Case Study 47: Autonomic Dysreflexia

CASE HISTORY

An 18-year-old teen athlete who was driving with other teens was involved in a motor vehicle crash, hitting a pole. He unfortunately sustained upper spinal cord injury at a T6 level. He is wheel chair bound and he has loss of bladder and bowel control. He uses straight catheterization of the bladder every 4–6 hours, for relief. All of a sudden the patient started having severe headache, blurring of vision and vomiting along with profuse sweating and the redness (erythema) in the upper extremities. His heart rate is 55 beats/minute and his blood pressure (BP) is 220/115 mm Hg.

DISCUSSION

Autonomic hyperreflexia is characterized by the sudden onset of headache and hypertension in a patient with a lesion above the T6 level. There may be associated bradycardia, sweating, dilated pupils, blurred vision, nasal stuffiness, flushing or piloerection. It usually occurs several months after the injury and has an incidence as high as 85% in quadriplegic patients. Frequently, it subsides within 3 years of injury, but it can recur at any time. Bowel and bladder distention are common causes. Hypertension is the major concern because of associated seizures and cerebral hemorrhage.

Autonomic hyperreflexia is associated with spinal cord injury patients (usually T6 or above) sometime after initial injury:
- Vasculature has adapted to loss of sympathetic tone
- BP normalized
- No vasodilation response to increased BP.

Autonomic nervous system reflexively responds with arteriolar spasm.
- Increased BP
- Stimulates peripheral nervous system (PNS)
- Results in bradycardia
- Peripheral and visceral vessels unable to dilate.

CLINICAL PRESENTATION

- Paroxysmal hypertension
- Headache
- Blurred vision
- Sweating and flushed skin above level of injury

- Increased nasal congestion
- Nausea
- Bradycardia
- Distended bladder or rectum.

Autonomic hyperreflexia is a reaction of the autonomic (involuntary) nervous system to overstimulation. This reaction may include high BP, change in heart rate, skin color changes (palor, redness, blue-gray skin color) and excessive sweating.

CAUSES

The most common cause of autonomic hyperreflexia is spinal cord injury (SCI) because the types of stimulation that are tolerated by healthy people can create an excessive response from the nervous system of a SCI patient.

Other causes include medication side effects, use of stimulants such as cocaine or amphetamines, Guillain–Barré syndrome, subarachnoid hemorrhage, severe head trauma and other brain injuries.

A number of conditions have symptoms as autonomic hyperreflexia, but have a different cause:

- Carcinoid syndrome is a disease caused by abnormalities of hormone-producing cells in the lungs and the gut.
- Neuroleptic malignant syndrome is a condition which causes muscle stiffness, high fever and drowsiness. This is caused by combinations of certain medications.
- Serotonin syndrome is caused by an abnormal release of serotonin from the brain.
- Thyroid storm is caused by production of too much thyroid hormone.

EXAMINATIONS AND TESTS

Signs

Signs often seen on examination include:

- Dilated pupils
- Flushed (red) skin above the level of SCI
- High BP
- Bradycardia or tachycardia.

Tests

Tests may include:

- Blood and urine tests
- Brain pictures including head computed tomography (CT) scan or magnetic resonance imaging (MRI)

- Electrocardiography (ECG)
- Lumbar puncture
- Spine pictures, particularly MRI
- Tilt-table testing
- Toxicology screening to look for stimulants
- X-ray.

TREATMENT

This condition is life-threatening, so it is important to quickly identify and treat the problem. A person with symptoms of autonomic hyperreflexia should sit up with their head raised and tight clothing should be removed.

Treatment will be based on the cause. If medications or drugs are causing the symptoms, the drugs must be stopped and any underlying illness must be treated.

If a slowed heart rate is causing the symptoms, anticholinergic medications may be helpful. Very high BP must be treated quickly but carefully. A sudden severe drop in BP can occur and can cause further issues. Commonly used emergency drugs for high BP include: nifedipine, nitroglycerin, phenoxybenzamine hydrochloride, mecamylamine and diazoxide.

A pacemaker may be needed for certain unstable heart-related situations.

PROGNOSIS

Prognosis is dependent on the underlying cause. If medications are the cause, patients usually recover with withdrawal of the medication. If other factors are the cause, prognosis depends on the successful treatment of the underlying condition.

COMPLICATIONS

Complications may occur as a result of medications used to treat the hyperreflexia, If the pulse rate drops severely, cardiac arrest can result.

Prolonged, severe high BP may cause seizures, occurlar hemorrhage, stroke or death.

PREVENTION

Prevention of autonomic hyperreflexia includes avoiding medications that cause this condition or make it worse. In SCI patients, there are a number of steps to avoid this complication including avoiding letting the bladder become too full, keeping pain levels low, and practicing proper bowel care to avoid stool impaction.

Fig. 1: Richard Marvin Hansen

HISTORICAL CASE

Richard Marvin Hansen is a famous Canadian athlete, who was the final torchbearer in the Winter Olympics of 2010 and also spoke during its opening ceremony. When he was 15, he was riding with his friend in a pickup truck and the truck swerved and hit a tree. From this accident, he received a spinal cord injury. Now he runs the Rick Hansen Foundation, a nonprofit foundation, which has generated more than $200 million for spinal cord injury-related programs. The goal is to effect the changes in clinical practice necessary to achieve the best possible outcome for victims of spinal cord injury (Fig. 1).

ADDITIONAL READING

1. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1743257
2. http://en.wikipedia.org/wiki/Rick_Hansen

CHAPTER

7

Endocrine

Case Study 48: Thyroid Storm

Remember the 3 P's and 2 S's in the management of thyroid storm.

—Badar M Zaheer

CASE HISTORY

History of Present Illness

A 47-year-old Navajo native American female is presented to the emergency department with her husband. She has chief complaint of fearfulness and a sudden onset of feeling as though her heart is going to explode out of her chest. She is also presented with difficulty in speaking due to a stiff jaw.

Past Medical History

She was suffering from hypertension since age of 40 years. She was being treated with hydrochlorothiazide (HCTZ) tablets 25 mg/day for the past 7 years with no renal organ involved.

Medications

- Daily HCTZ, PO, 25 mg in the morning since the age of 40 years, for 7 years.
- Herbal supplements.

PHYSICAL EXAMINATION

General Appearance

Patient is apprehensive, delirious, obtunded, anxious, in distress, restless, and irritable to staff and family members. Patient vomits and has diarrhea several times during the encounter.

Vital Signs

Her vital signs are temperature 104°F, blood pressure 210/110 mm Hg, heart rate 180 beats/minute, respiratory rate 30 breaths/minute, oxygen

saturation 99%, height 66 inches, weight 160 pounds and body mass index 25.8 kg/m^2.

Head and Neck

- No evidence of trauma, tumor, facial edema, carotid bruits or thyroid nodules.
- Diffuse goiter seen.
- *Mental status*: Delirious, obtunded, anxious and irritable. No head injury.
- *Pupillary size*: 3 mm, symmetric, reactive to light and accommodation. Sclera is icteric with dry oral mucous.
- Patient has rigidity of the jaw, and has difficulty and hesitancy in articulate due to discomfort.
- Fine silky hair.

Cardiac Examination

- Point of maximal impulse is palpated at the left 5th intercostal space at the point of intersection with the left midclavicular line.
- Heart auscultated at apex and base tachycardic, irregular rhythm and forceful impulse.
- *Heart sounds*: S4 gallop, S1, S2 and prominent systolic flow murmur.

Pulmonary Examination

- No wheezes, rales or rhonchi.
- *No evidence of consolidation*: No bronchial breath sounds or egophony.
- *No increased work of breathing*: No retraction, no accessory muscle use
- Abdominal breathing and tachypnea.

Abdominal Examination

Negative

Neurologic Examination

- Obtunded, apprehensive, irritable and anxious.
- Intact cranial nerves II–XII; difficulty in opening jaw to test masseter muscle is noted.
- Sensation, reflex, cerebellar function found intact.

Laboratory Tests

- *Thyroid*: Free T4 levels are elevated and thyroid stimulating hormone (TSH) levels are reduced.
- CBC and BMP are within normal limits.
- EKG shows sinus tachycardia.

CASE DISCUSSION

- Normal temperature in the context of hypothyroidism may indicate a comorbid infection.
- If you suspect thyroid storm with congestive heart failure, always obtain an echocardiogram prior to administering beta blockers. If the echo shows the CHF is secondary to high-ouput failure, you can still use beta blockers with caution.

IMPRESSION

Thyroid storm: Patient seemed to have had hyperthyroidism that did not receive medical attention and the recent upper respiratory infection has precipitated the condition.

DIFFERENTIAL DIAGNOSIS

- Hypertensive crisis
- Hypertensive emergency
- Acute pulmonary edema
- Heat stroke
- Malignant hyperthermia
- Sepsis/septic shock
- Sympathomimetic toxicity
- Tachyarrhythmia
- Pheochromocytoma.

TREATMENT

Supportive Therapy

- IV normal saline, 200 cc/hour
- IV dextrose 5% in 0.45% saline, 200 cc/hour
- IV Glucocorticoids (steroids) are given
- Oxygen is provided, cooling blankets are used.
- Management can be summarized by remembering the 3 P's and 2 S's.

P—Propranolol to reduce beta-adrenergic symptoms and quell peripheral conversion of T4 to T3

P—Propylthiouracil to reduce thyroid hormone synthesis and also assist propranolol in decreasing peripheral conversion of T4 to T3

P—Potassium iodide to reduce hormone release

S—Steroids to decrease peripheral T4 to T3 conversion

S—Supportive care

Medication

- Propylthiouracil (PTU) × 100 mg/2 hour (antithyroid drug).
- Iodine (inhibit thyroid hormone release).
- Propranolol, 40 mg IV push (to control tachycardia) is given.
- Dexamethasone IV 4 mg × 3 (inhibit prostaglandin H release; impair peripheral T4 to T3 conversion; provide adrenal support).

FOLLOW-UP

Patient was transferred to another hospital after being stabilized, and a follow-up call confirmed that the patient survived and was transferred to a medical floor after being 24 hours in the intensive care unit.

Further follow-up indicates antithyroid agent was stopped 10 days prior to treatment and following radioactive iodine treatment. She was then stabilized and put on daily levothyroxine after radioactive ablation therapy. She is scheduled for a full physical examination in 2 weeks.

Case Study 49: Diabetic Ketoacidosis

CASE HISTORY

Basic Information

Patient is a 15-year-old Caucasian male.

History of Present Illness

A 15-year-old confused white male is brought to the emergency room (ER) on Friday night by his parents who state that the patient suffers from anorexia, profound nausea, repeated vomiting, epigastric pain and shortness of breath for almost 24 hours. Parents report starting early this week because patient had an incredible increase in appetite and complained of extreme thirst and increased volume of urination during the night.

The patient looks direly ill as he enters the emergency room; his skin and mucous membranes look extremely dry. His respirations are laboured. He has decreased skin tugor, decreased reflexes, and characteristic acetone like breath odor.

Lab Results:

Glucose: 850
Sodium: 125
Potassium: 4.5
Bicarbonate: 5
BUN: 30
PH: 7.1
PCO_2 (mm Hg) 17

CASE DISCUSSION

This clinical presentation is typical of Diabetic Ketoacidosis. It is a metabolic emergency. Delay in treatment may result increase morbility and mortality. DKA may present as an initial presentation of Type 1 Diabetes in 25% of cases. Majority of the Case are Type 1 Diabetes but some patients with Type 2 Diabetes may also present with DKA under severe physiologic distress. Most of the patients will present with the classical symptoms of polyuria, polydipsia, polyphagia, and fatigue. Other may present with respiratory difficulty because of metabolic acidosis. Some others may present with idiopathic symptoms such as severe abdominal pain. The precipitating factors are usually infection, stress,

travel, dehydration. Some patients may present with altered sensorium and history is unobtainable. The hallmark of DKA is a combination of hyperglycemia, elevated anion gap, and serum bicarbonate level below 20 mEq/L. Usually there is no correlation between the severity of hyperglycemia and the severity of ketoacidosis. Ketones in the serum and the blood and urine will be positive.

MANAGEMENT OF DKA

As the name itself indicates, DKA is diabetes (hyperglycemia+ ketosis+ acidosis (metabolic). Except in the rare cases of anuric patient, the absense of ketones in the urine reliably excludes the diagnosis of DKA. Patients in DKA can have significant fluid deficits, sometimes upto 5-10 L. Shock is fairly common, and must be managed right away with crystaloid infusion to prevent organ damage. Adults with clinical shock should receive an initial 2 L bolus of normal saline with frequent reassessments. In children, shock is treated with 20 mL/kg of normal saline. Although there is a possibility of over-hydrating that can present substantial complications later in the course of treatment, rectifying shock is a more concern. If shock is not treated right away, it will contribute to severe acidosis.

Insulin

- 0.1 unit/kg IV push, then 1 L/kg/hr by continuous infusion. Decrease dose rate 50% when serum HCO3 rises above 16 mEq/L.
- Do not try intramuscular injections which are painful and less reliably absorbed when the patient is in shock. The combination of treatment of rehydration and insulin will usually lower serum glucose much faster than ketones are cleared. In any event, insulin infusion should continue until the anion gap returns to normal. When the serum glucose falls between 200-300 mg/dL (11,1–16.7 mmol/L), dextrose infusion should be added to prevent hypoglycemia. Insulin binds to IV tubing, so a thorough flush with a drip solution is necessary.

Fluids

- Start with 0.9% NS, 1L/hr for the first 2 hours. Follow with 0.45% saline at 250-500 mL/hr. Total fluid deficit is usually 50-100 mL/kg. Simply reversing shock with normal saline and then infusion of half normal saline at 2–3x the maintenance dose is usually sufficient.

Potassium

- Usually the potassium deficit is quite large. But the serum potassium may still show low, normal, or even high. If potassium is initially

elevated, look and treat for hyperkalemia based on the EKG findings. You can continue giving fluids without potassium until the serum potassium returns normal.

If the initial potassium is normal or low, potassium can be given immediately. Magnesium supplementation may be necessary sometimes to help the patient to retain potassium.

Phosphate

- Usually phosphate supplementation is not required and has little impact. But, if the phosphate depletion is severe enough where it falls below 1 mg/dL, then it may be necessary for replacement therapy. Recommended dose is 7.7 mg/kg over 4 hours.

Sodium Bicarbonate

- Causes more harm than good. Supplementation is not Recommended regardless of the severity.

Pathophysiology

- Severe insulin insufficiency resulting in decreased glucose uptake leading to hyperglycemia and osmotic diuresis which results in electrolyte depletion, dehydration and acidosis.
- *Increased proteolysis*: Increased nitrogen loss and increase in amino acids leading to gluconeogenesis and glycogenolysis resulting in hyperglycemia.
- Lipolysis leading to increase in glycerol and free fatty acid levels.
    - Glycerol contributing to gluconeogenesis and glycogenolysis.
    - Increased free fatty acids cause ketogenesis which leads to ketonemia, then ketonuria and finally, acidosis.

Findings in DKA

- Leukocytosis without toxic granulations, secondary to release of stress hormones.
- Hemoconcentration secondary to dehydration, if hematocrit (HCT) less than 35% of suspected blood loss.
- Serum amylase elevation in 40–80% cases; no relation to severity, morbidity, or mortality.
- *Renal amylase/creatinine clearance ratio*: Raised without clinical symptoms of acute pancreatitis.
- *Serum lipase*: Normal.
- Abnormal values for SGOT, SGPT, lactate dehydrogenase (LDH) and other liver enzymes in 33% cases.

- No correlation between serum hepatic enzymes levels and severity of abdominal symptoms.
- Reversible hepatocellular damage, sufficient to allow release of cytosolic enzymes.
- Fatty infiltration of liver and reduced hepatic perfusion may contribute.

Case Study 50: Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)

CASE HISTORY

A 70-year-old male is presented to the emergency department (ED) with significant lethargy. Patient is unable to answer questions but family informs that recently he has lost a lot of weight and has not been eating. He has been coughing more than usual and the sputum occasionally contains blood. They have been trying to get him to see a doctor, but he has been refusing. This morning when his son went to visit him at his home where he normally lives by himself, his son found him in this confused state and immediately called emergency medical services (EMS). Patient is minimally responsive and oriented only to person. Pupils are equal and reactive. Patient withdraws to pain in all four extremities. Patient is afebrile, and his blood pressure is 100/75 mm Hg, pulse rate 101 beats/minute, respiratory rate 22 breaths/minute and oxygen saturation level remained 93% on room air with administration of 2 L oxygen via nasal cannula.

Laboratory Test

- $Na^+ \downarrow$ 120
- K^+ 4.7 mEq/L
- Bicarbonate (HCO^3) 35mEq/L
- Serum Osmolality 265 mOsm/Kg (Normal range 275–290)
- Urine Osmolality 390 mOsm/kg (normal range > 800 mOsm/kg

DISCUSSION

The syndrome of inappropriate antidiuretic hormone (ADH) secretion (SIADH) is a syndrome of hyponatremia due to inappropriate or increased secretion of ADH wherein water is retained causing hyponatremia, concentrated urine more than 100 mOsm/L in the face of hypotonic plasma less than 260 mOsm/L. The key to understanding the pathophysiology, signs, symptoms, and treatment of SIADH is the awareness that the hyponatremia is a result of an excess of water rather than a deficiency of sodium.

SIGNS AND SYMPTOMS

Anorexia, nausea, headache, vomiting, altered mental status and seizure activity may present in acute cases depending on the severity

of hyponatremia like confusion, disorientation, delirium, generalized muscle weakness, myoclonus, tremor, asterixis, hyporeflexia, ataxia, dysarthria, Cheyne-Stokes respiration, generalized seizures and coma. Chronic hyponatremia rarely causes any acute symptoms. Symptoms may not correlate with the severity of the condition.

DIAGNOSIS

In the absence of a single laboratory test to confirm the diagnosis, SIADH is best defined by the classic Bartter-Schwartz criteria, which can be summarized as follows:

- Hyponatremia with corresponding hypo-osmolality
- Continues renal exertion of sodium
- Urine less than maximally dilute
- Absence of clinical evidence of volume depletion
- Absence of other causes of hyponatremia
- Correction of hyponatremia by fluid restriction.

The patients volume should be assessed clinically to help rule out the presence of hypovolemia. Imaging studies that may be considered include the following:

- Chest radiography (for detection of an underlying pulmonary cause of SIADH, like Small Cell Carcinoma of lung).
- Computed tomography or magnetic resonance imaging of the head (for detection of cerebral edema occurring as a complication of SIADH, for identification of a CNS disorder responsible for SIADH, or for helping to rule out other potential causes of a change in neurologic status).

MANAGEMENT

Treatment of SIADH and the rapidity of correction of hyponatremia depend on the following:

- Degree of hyponatremia

Mild–Only water restriction less than 500 mL per day is enough and vasopressin-2 receptor antagonist if needed.

If the sodium level is below 115 mEq/L associated with seizure 3% normal saline 1 mL/kg/h to raise the sodium concentration by not more than 2 mEq/L/h and not more than 10–12 mEq/L/h in the first 24 hours. to avoid central pontine myelinolysis (CPM).

If the duration of hyponatremia is unknown and the patient is asymptomatic, it is reasonable to presume chronic SIADH. In an emergency setting, aggressive treatment of hyponatremia should always be weighed against the risk of including central pontine myelinolysis (CPM). Such treatment is warranted as follows:

Second-line drugs furosemide 40 mg IV and demeclocycline 300–600 mg.

ADDITIONAL READING

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Case Study 51: Adrenal Crisis

CASE HISTORY

A 45-year-old man is brought to the emergency department (ED) by his wife with chief complaints of confusion and delirium. He's also been having nausea, vomiting, weight loss and looks extremely dehydrated. He had symptoms of upper respiratory tract infection preceding this ED visit as described by his wife. Physical examination findings include a temperature of 98°F, blood pressure (BP) 68/40 mm Hg, heart rate 140 beats/minute, serum glucose of 34 mg/dL and hyperpigmented skin.

ASSESSMENT

The patient is experiencing acute adrenal insufficiency. Low serum glucose and BP are a result of low levels of glucocorticoids. The presence of hyperpigmentation of the skin is indicative of high serum levels of adrenocorticotropic hormone (ACTH) and thus primary adrenal insufficiency.

DIAGNOSTIC TEST IN THE ER

Cosyntropin (synthetic ACTH) stimulation test.

A baseline cortisol level is drawn. 0.25 mg of cosyntropin is given IM or IV. After one hour a repeat cortisol level is drawn which should be double the baseline level. If there is no increase in the cortisol level, it means it is primary adrenal insufficiency.

EMERGENCY ROOM CARE AND DISPOSITION

The patient underwent fluid resuscitation with rapid administration of 2 L of normal saline. 50 mL of 50% dextrose was administered via intravenous (IV) push and hydrocortisone 300 mg IV. Patient subsequently became more awake, alert and stable. On examination, BP was 105/50 mm Hg, serum glucose 72 mg/dL, serum sodium (Na^+) 124 mEq/L and serum potassium (K^+) 5.5 mEq/L

The goal of the treatment is to replace crystallite fluids, glucocorticoids and mineralocorticoids. To correct volume, glucose and sodium deficits is our object.

Hydrocortisone 100–300 mg IV every 6–8 hours is sufficient to provide glucocorticoids and mineralocorticoids.

DISCUSSION

Adrenal crisis is a life threatening emergency of adrenal insufficiency. They usually present in a state of shock or change in mental status.

The adrenal cortex of the adrenal glands produces a number of steroid hormones: Aldosterone, glucocorticoids and androgen hormones. Aldosterone is responsible for maintaining BP and to excrete excess serum K^+. Its secretion is stimulated by the renin-angiotensin pathway and high serum K^+. Glucocorticoids are responsible for maintaining serum glucose, potentiating vasoconstriction, inhibiting inflammatory responses, and are associated with memory formation and arousal. Glucocorticoid production is controlled by the hypothalamus-anterior pituitary-ACTH pathway. Androgens are responsible for production of sex hormones (including testosterone and estrogen) and are responsible for sexual development and other complex functions.

Aldosterone and glucocorticoids are crucial to metabolism, BP control, and electrolyte balance and their absence is incompatible with life. For this reason, adrenal insufficiency will present with symptoms related to hormonal functions and must be quickly recognized.

Adrenal insufficiency is characterized by underproduction of glucocorticoids. Glucocorticoids maintain serum glucose by stimulating gluconeogenesis, fatty acid metabolism and amino acids breakdown. They also inhibit insulin sensitivity and thus the uptake of glucose, hence further increases serum glucose. Glucocorticoids also maintain BP in conjunction with the sympathetic system by potentiating the vasoconstrictive effects of epinephrine and norepinephrine on blood vessels. When there are insufficient glucocorticoids, low serum glucose, hypotension, and reflex tachycardia are expected.

Glucocorticoids production is stimulated by ACTH, which is secreted by the anterior pituitary under the influence of hypothalamus. Serum ACTH therefore provides us with clues to the etiology of glucocorticoid insufficiency. In primary adrenal insufficiency, the adrenal glands themselves are the origin of the problem. Because hypothalamic-pituitary-adrenal axis is intact, the lack of negative-feedback by glucocorticoids stimulates ACTH production, which translates into high circulating ACTH. Moreover, because ACTH shares homology with melanocyte stimulating hormone (MSH), the patients often present with hyperpigmentation of the skin. Primary adrenal insufficiency can arise from gradual destruction of the glands. For example, Addison's disease is characterized by autoimmune destruction of the adrenal glands. Tuberculosis (TB) can disseminate to the glands. While rare in the United States, TB is the leading cause of adrenal insufficiency in third-world

countries. Because these diseases tend to be chronic, prolonged excessive ACTH may lead to hyperpigmentation. Primary adrenal insufficiency can also occur acutely. For example, Waterhouse-Friderichsen syndrome is the hemorrhage of the adrenal glands following a meningococcal septicemia, or disseminated intravascular coagulation (DIC). In this case, hyperpigmentation is not observed as ACTH elevation is not prolonged.

In contrast, secondary adrenal insufficiency is caused by deficiency in ACTH. This may arise from lesions in either hypothalamus [lack of corticotropin-releasing hormone (CRH)] or pituitary. Motor vehicle accidents, Sheehan's syndrome and tumor are just some potential causes of secondary adrenal insufficiency.

However, the most common cause of acute secondary adrenal insufficiency in the Western world is chronic exogenous steroids. Chronic use of synthetic steroids depresses ACTH secretion, thus decreasing the stimulus needed for glucocorticoid production. If the patient suddenly withdraws from steroid medications, the adrenal glands are unable to ramp up the production to compensate for the sudden drop of glucocorticoids. This sudden adrenal insufficiency or adrenal crisis can present with symptoms as described by the case history. To avoid adrenal insufficiency secondary to chronic steroid use, clinicians need to gradually taper down the dosage of patients' steroid medications before completely stopping steroid therapy.

Because adrenal cortex also produces aldosterone, aldosterone deficiency and associated hyperkalemia can occur in some forms of adrenal insufficiency. In Addison's disease and Waterhouse-Friderichsen syndrome, the entire adrenal cortex is damaged. Therefore, one can expect symptoms of both glucocorticoid and aldosterone deficiency. On the other hand, secondary adrenal insufficiency is due to lack of ACTH, which mainly controls the production of glucocorticoids. As a result, only isolated glucocorticoid deficiency is manifested.

CHAPTER

8

Psychiatric Emergencies

Case Study 52: Panic Attack

CASE HISTORY

A 46-year-old male with no history of hypertension or high cholesterol is presented to the emergency room (ER). The patient's chief complaint is chest heaviness and left arm pain that started early in the morning. He also complained of having shortness of breath at home. These symptoms lasted approximately 2 hours. When they did not subside, he became very panicky. He started to breath heavily, became lightheaded, and also had tingling around his mouth and fingertips. At this point, the patient felt like he was going to die and felt he should go to the ER. He presented to the emergency department (ED) with mild chest heaviness and pain that started again though his arm, neck, jaw, and back pain had gone away. The patient did not complain of nausea or emesis. He has been a half-pack per day smoker for the last 10 years. The patient's father had a heart attack when he was 55 years old. The patient eats spicy food and has a history of reflux. The patient does not have any headache, focal numbness, tingling and weakness at time of presentation. He reports taking one baby aspirin at home earlier today. Serial electrocardiography (ECG) is done three times and shows normal sinus rhythm. Normal cardiac enzymes, creatine kinase (CK), creatine kinase-muscle and brain (CK-MB) and troponin, are all within normal limits. The values of brain natriuretic peptide and D-dimer are also normal.

DISCUSSION

Panic attack is a diagnosis of exclusion because the symptoms and signs mimic those of several potentially life threatening emergency conditions like MI (Myocardial infarction) and PE (Pulmonary embolism).They are characterized by a surge of intense fear or discomfort. The fear reaches its climax very quickly (usually within minutes). While the patient is

experiencing this fear, four or more of the following symptoms must also be observed within 10 minutes [according to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM)-5]:[1]

1. Palpitations, pounding heart or accelerated heart rate
2. Sweating
3. Trembling or shaking
4. Sensations of shortness of breath or smothering
5. Feeling of choking
6. Chest pain or discomfort
7. Nausea or abdominal distress
8. Feeling dizzy, unsteady, lightheaded or faint
9. Derealization (feelings of unreality) or depersonalization (being detached from oneself)
10. Fear of losing control or going crazy
11. Fear of dying
12. Paresthesias (numbness or tingling sensations)
13. Chills or hot flashes.

A panic attack that comes into the ER and is not stabilized or resolved often results in panic disorder. Panic disorder falls under the heading of anxiety disorders, which by definition are mental illnesses characterized by abnormal, pathological fear and anxiety. The DSM-5 definition for diagnosing panic disorder is as follows:[2]

(A) Both (1) and (2):

1. Recurrent unexpected panic attacks
2. At least one of the attacks has been followed by 1 month (or more) of one (or more) of the following:
   (a) Persistent concern about having additional attacks.
   (b) Worry about the implications of the attack or its consequences (e.g. losing control, having a heart attack, "going crazy").
   (c) A significant change in behavior related to the attacks.

(B) The presence (or absence) of agoraphobia.

(C) The panic attacks are not due to the direct physiological effects of a substance (e.g. a drug abuse, a medication) or a general medical condition (e.g. hyperthyroidism).

(D) The panic attacks are not accounted for by other mental disorders, such as social phobia (e.g. occurring on exposure to feared social situations), specific phobia (e.g. on exposure to a specific phobic situation), obsessive-compulsive disorder (e.g. on exposure to dirt in someone with an obsession about contamination), posttraumatic stress disorder (e.g. in response to stimuli associated with a severe stressor) or separation anxiety disorder (e.g. in response to being away from home or close relatives).

When an ED doctor sees people like in the case above, the immediate response may be that the patient is having a heart attack. We can never assume that it is panic attack unless everything is ruled out. The initial treatment given to this patient is three baby aspirin and 1 inch of nitroglycerine paste. The patient is admitted to rule out coronary syndrome, and only after the doctor had a chance to look at the chart did the symptoms appear to be more consistent with panic attack.

TREATMENT

- First exclude life threatening conditions.
- Benzodiazepines as discussed below.

If the patient is in an acute episode, antianxiety medications (Xanax, Klonopin, Ativan) may help to alleviate symptoms and sedate the patient. Quick-acting benzodiazepines (lorazepam, alprazolam, clonazepam) may be used in addition to active support and assurance of safety.[3] The typical dose and route is 0.5 mg lorazepam given intravenously every 20 minutes until symptoms have subsided. The physician should remember to talk in a slow calm voice, and sit down with the patient to help calm them down. A psychiatric referral is often given after the patient's acute symptoms have been relieved.

REFERENCES

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Case Study 53: Psychosis

"One in every eight ER visits in 2007 involved either a diagnosis of a mental health or substance abuse condition"
—Agency for Healthcare Research and Quality

CASE HISTORY

A 60-year-old man who is a known, successful restaurant owner is brought to the emergency department by his family members for being isolated and suspicious of his wife having extramarital relations with another man. The man is suspecting his son to be a murderer, and the man thinks his son will murder him. He does not trust any of his family members, he thinks they are stealing his property. The man often exclaims to his wife that "God has sent me here, and he told me you are stealing from me".

DISCUSSION

Psychosis is typically seen as drastic changes in personality and a loss of contact with reality. This condition which commonly appears as the defining symptom of many mental disorders (schizophrenia, schizo-affective, delusional) is characterized by delusions and hallucinations. Auditory hallucinations are most commonly associated with phyctriatic causes of psychosis whereas visual hallucinations have organic cause. Impaired functioning and distorted view of reality may also be seen with classical cases of psychosis. Psychosis may be caused by psychological, social and biological factors; the trick is figuring out which one is the culprit. Episodes may be caused by a variety of things not limited to: alcohol, illegal drugs, brain tumor, some steroids, epilepsy, schizophrenia, and genetic abnormalities. Another possible factor that influences psychosis is migration. It has been found that people immigrating to other countries often have higher incidence of psychosis than natives.

When diagnosing psychosis, the physician should first rule out delirium, noting that psychosis should never have fluctuating consciousness or reduced clarity of awareness.[1] If biological and social factors are ruled out, diagnosis should be followed by a referral for psychological consultation to determine if another mental disorder is responsible.

Our ED physicians have limited resources on the evaluation and treatment of patients with mental health issues. American Association for Emergency Physciatry (AAEP) is providing an online help for ER physicians. It may not be useful as a real time quick reference guide, but nevertheless, it provides guidelines for physciatric emergencies. Modified SAD PERSONS scale, a clinical decision making tool, can

evaluate the risk of suicide of a patient. For assessing the suicidal risk at the time of discharging a patient, this 10 question tool can be easily used by the ER physician. As the patient population in the emergency room (ER) for mental health condition increases, there is a greater need for developing a more standardized and a robust emergency residency program to treat and provide a proper treatment for patients with mental health issues.

TREATMENT

Note: Before patients can receive antipsychotic medications, blood work must be drawn. Tests include fasting lipid profile, fasting blood sugars, liver function tests, metabolic panel and weight.

Atypical antipsychotic medications are the classic treatment for psychotic patients because they decrease the risk of extrapyramidal symptoms while still treating the patient. Specifically when using olanzapine and clozapine there is less risk of hyperlipidemia, new-onset diabetes or weight gain.1 Typical dosing for olanzapine is 5–10 mg intramuscular (IM) with up to 3 × 10 mg injections over a 24-hour period. Also, the physician may order benzodiazepines such as ziprasidone 5–20 mg IM every 4–6 hours with a maximum of 40 mg in a 24-hour period.

Lastly, the physician must remember that a person who is diagnosed with psychosis or having an acute psychotic episode may be legally hospitalized against their will. In particular, if this patient is violent or threatening harm to themselves or others, especially if they plan to commit suicide. Also, the physician may hospitalize a patient that is malnourished or ill because they fail to feed themselves. Finally, the physician must look at things like dress being appropriate for the climate when doing intake on a patient to see if they are able to care for themselves or not.

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ADDITIONAL READING

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CHAPTER

9

Environmental Injuries, Toxicology and Animal Bites

Case Study 54: Peanut Anaphylaxis

"7th Grader Dies at School Event from Food Allergy." —CBS News 2011

CASE HISTORY

Chief Complaint: Allergic Reaction

A 13-year-old girl was rushed to the emergency department (ED) from a school event. While sitting with a group of friends and eating Chinese food, she became a vibrant red and was having difficulty breathing. She collapsed moments after and was unable to be resuscitated. At the ED, she had a feeble pulse and no breath sounds. The blood pressure was not palpable. Intravenous (IV) lines were placed and patient was immediately intubated. Fluids, epinephrine and Benadryl were all given.

Soon afterwards the patient lost her pulse and her blood pressure never recovered. Resuscitation attempts were unsuccessful. The patient passed away a few hours after the initial presentation.

Her family denied any previous medical history. Her mother had an uneventful course in pregnancy. She was born at full term, appropriate for gestational age (AGA) with no malformations. Her parents report that she had a peanut allergy. The school officials report that they were aware of the allergy and made sure that the Chinese food they ordered had no peanuts.

Allergies

None known at time of presentation.

PHYSICAL EXAMINATION

- *Skin*: Urticaria, skin flushing
- *Vitals*: Blood pressure (BP) was not measurable
- *Cardiovascular system*: Weak, decreased heart sounds
- *Respiratory system*: No breath sounds on initial presentation.

DIAGNOSIS

Patient has anaphylactic reaction due to peanut allergy. While the school ensures that the Chinese food ordered did not have any peanuts, many Chinese restaurants use peanut oil without considering it as a peanut product.

Differential Diagnosis

- *Esophageal*: Globus hystericus, hereditary angioedema, foreign body aspiration (young children, especially) and angioedema.
- *Endocrine*: Pheochromocytoma and malignant carcinoid syndrome.
- *Hematology/Oncology*: Mastocytosis, thyroid and medullary carcinoma.
- *Cardiac*: Shock, capillary leak syndrome and myocardial dysfunction.
- *Respiratory*: Pulmonary embolism.
- *Neurological*: Vasodepressor (vasovagal) reaction (probably the most common masquerader) and autonomic epilepsy.
- *Psychiatric*: Panic attacks and vocal cord dysfunction syndrome.
- *Toxic*: Scombroid fish poisoning, monosodium glutamate poisoning, Red man's syndrome and ethanol toxicity.

ANAPHYLAXIS

Pathophysiology

Anaphylaxis is considered as a Type I/immunoglobulin E (IgE)-mediated allergic reaction. Sudden activation of mast cells occurs by IgE antibodies when exposed to an allergen. The mast cells then release inflammatory mediators. The physiologic responses to the release of anaphylaxis mediators include smooth muscle spasm in the respiratory and gastrointestinal (GI) tracts, vasodilation, increased vascular permeability and stimulation of sensory nerve endings. Increased mucous secretion and increased bronchial smooth muscle tone, as well as airway edema, contribute to the respiratory symptoms observed in anaphylaxis. Anaphylactoid reactions have an identical clinical manifestation that results from mast cell degranulation without IgE mediation.

The frequency of anaphylaxis is increasing, and this has been attributed to the either an increased number of allergens that people are exposed to or a sterile lifestyle during childhood development. Evidence of the former is provided by the increased correlation between the rising rate of anaphylaxis and increased pollution. The lifetime prevalence of anaphylaxis is 1–2% of the population. The frequency of anaphylaxis is increasing, and this has been attributed to the increased number of potential allergens to which people are exposed.

Up to 500–1,000 fatal cases of anaphylaxis per year are estimated to occur in the United States. Estimated mortality rates range from 0.65–2% of patients with anaphylaxis.

Immunologic IgE-Mediated Reactions

Certain foods are more likely than others to elicit an IgE antibody response and lead to anaphylaxis. Foods likely to elicit an IgE antibody response in all age groups include: peanuts, tree nuts, fish and shellfish. Those likely to elicit an IgE antibody response in children also include eggs, soy and milk.

An analysis of 32 fatalities thought to be due to food-induced anaphylaxis revealed that peanuts likely were the responsible food in 62% of the cases. In placebo-controlled food challenges, peanut-sensitive patients can react to as little as 100 μg of peanut protein.[1] The Rochester epidemiology project, in agreement with earlier studies, found that food ingestion was the leading cause of anaphylaxis, accounting for as many as one-third of all cases.

Scombroid fish poisoning can occasionally mimic food-induced anaphylaxis. Bacteria in spoiled fish produce enzymes capable of decarboxylating histidine to produce biogenic amines, including histamine and cis-urocanic acid, which is also capable of mast cell degranulation.

Immunologic IgE-Independent Reactions

Anaphylaxis may result from administration of blood products including IV immunoglobulin, or animal antiserum, at least partly as a consequence of activation of the complement cascade. Certain byproducts of the cascade are capable of causing mast cell/basophil degranulation.

Exercise-induced anaphylaxis is a rare syndrome that can take one of two forms. The first form is ingestant dependent, requiring exercise and ingestion of particular types of food (e.g. wheat, celery) or medications [e.g. nonsteroidal anti-inflammatory drugs (NSAIDs)] to cause an episode of anaphylaxis. In these patients, exercise alone does not produce an episode, and ingesting the culprit food or medication alone does not cause an episode.

The second form is characterized by intermittent episodes of anaphylaxis during exercise, independent of any food ingestion. Anaphylaxis does not necessarily occur during every episode of physical exertion.

Anaphylaxis can be a manifestation of systemic mastocytosis, a disease characterized by excessive mast cell numbers in multiple organs. Such patients appear to be at increased risk for food and venom reactions. Alcohol, vancomycin, opioids, radiocontrast media and other biologic agents that can degranulate mast cells directly are discouraged.

Nonimmunologic Reactions

Certain agents, including opioids, dextrans, protamine and vancomycin, are thought to cause direct, nonimmunologic release of mediators from mast cells. Evidence also exists that dextrans and protamine can activate several inflammatory pathways, including complement, coagulation and vasoactive (kallikrein-kinin) systems.

Intravenously administered radiocontrast media causes an anaphylactoid reaction that is clinically similar to true anaphylaxis and is treated in the same way. The reaction is not related to prior exposure. Approximately 1–3% of patients who receive hyperosmolar IV contrast experience a reaction. Reactions to radiocontrast media usually are mild (most commonly, urticarial), with only rare fatalities reported. Risk of a fatal reaction has been estimated at 0.9 cases per 100,000 exposures.

Pretreatment with antihistamines or corticosteroids and use of low-molecular weight (LMW) contrast agents lead to lower rates of anaphylactoid reactions to IV radiocontrast media (approximately 0.5%). Consider these measures for patients who have prior history of reaction, since rate of recurrence is estimated at 17–60%. Some institutions use only LMW agents. Personnel, medications and equipment needed for treatment of allergic reactions always should be available when these agents are administered. Obtain consent before administration.

Patients who are atopic and/or asthmatic are also at increased risk for reaction. In addition, allergic reaction is more difficult to treat in those taking beta-blockers.

Shellfish or iodine allergy is not a contraindication to the use of IV contrast and does not mandate a pretreatment regimen. As with any allergic patient, give consideration to the use of LMW contrast agents. In fact, the term "iodine allergy" is a misnomer. Iodine is an essential trace element present throughout the body. No one is allergic to iodine. Patients who report iodine allergy usually have had either a prior contrast reaction, a shellfish allergy, or a contact reaction to povidone-iodine (betadine).

Mucosal exposure [e.g. GI, genitourinary (GU)] to radiocontrast agents has not been reported to cause anaphylaxis; therefore, a history of prior reaction is not a contraindication to GI or GU use of these agents.

WORKUP

Presentation

Patients often describe a sense of impending doom, accompanied by pruritus and flushing. This can evolve rapidly into the following symptoms, broken down by organ system:

- *Cutaneous/Ocular*: Flushing, urticaria, angioedema, cutaneous and/or conjunctival pruritus, warmth and swelling.
- *Respiratory*: Nasal congestion, rhinorrhea, throat tightness, wheezing, shortness of breath, cough and hoarseness.
- *Cardiovascular*: Dizziness, weakness, syncope, chest pain and palpitations.
- *Gastrointestinal*: Dysphagia, nausea, vomiting, diarrhea, bloating and cramps.
- *Neurologic*: Headache, dizziness, blurred vision, and seizure (very rare and often associated with hypotension).
- *Others*: Metallic taste and feeling of impending doom.

PHYSICAL EXAMINATION

The first priority in the physical examination should be to assess the patient's airway, breathing, circulation and adequacy of mentation (e.g. alertness, orientation and coherence of thought).

General appearance and vital signs vary according to the severity of the anaphylactic episode and the organ system(s) affected. Vital signs may be normal or significantly disordered with tachypnea, tachycardia, and/or hypotension.

Patients commonly are restless due to severe pruritus from urticaria. Anxiety, tremor, and a sensation of cold may result from compensatory endogenous catecholamine release. Anxiety is common, unless hypotension or hypoxia causes obtundation. Frank cardiovascular collapse or respiratory arrest may occur in severe cases.

Respiratory Findings

Severe angioedema of the tongue and lips [as may occur with the use of angiotensin-converting enzyme (ACE) inhibitors] may obstruct airflow. Laryngeal edema may manifest as stridor or severe air hunger. Loss of voice, hoarseness, and/or dysphonia may occur. Bronchospasm, airway edema, and mucus hypersecretion may manifest as wheezing. In the surgical setting, increased pressure of ventilation can be the only manifestation of bronchospasm. Complete airway obstruction is the most common cause of death in anaphylaxis.

Cardiovascular Findings

Tachycardia is present in one-fourth of patients, usually as a compensatory response to reduced intravascular volume or to stress from compensatory catecholamine release.

Bradycardia, in contrast, is more suggestive of a vasodepressor (vasovagal) reaction. Although tachycardia is the rule, bradycardia has also been observed in anaphylaxis (see Pathophysiology). Thus, bradycardia may not be as useful for distinguishing anaphylaxis from a vasodepressor reaction as was previously thought. Relative bradycardia (initial tachycardia followed by diminished heart rate despite worsening hypotension) has been reported previously in experimental settings of insect sting anaphylaxis, as well as in trauma patients.[2]

Hypotension (and resultant loss of consciousness) may be observed secondary to capillary leak, vasodilation and hypoxic myocardial depression. Cardiovascular collapse and shock can occur immediately, without any other findings. This is an especially important consideration in the surgical setting. Because shock may develop without prominent skin manifestations or history of exposure, anaphylaxis is part of the differential diagnosis for patients who present with shock and no obvious cause.

Cognitive Findings

If hypoperfusion or hypoxia occurs, it can cause altered mental status. The patient may exhibit a depressed level of consciousness or may be agitated and/or combative.

Cutaneous Findings

The classic skin manifestation is urticaria (i.e. hives). Urticaria can occur anywhere on the body, often localizing to the superficial dermal layers of the palms, soles and inner thighs. Lesions are red and raised, and sometimes have central blanching. Intense pruritus occurs with the lesions. Lesion borders are usually irregular and sizes vary markedly. Only a few small or large lesions may become confluent, forming giant urticaria. At times, the entire dermis is involved with diffuse erythema and edema.

In a local reaction, lesions occur near the site of a cutaneous exposure (e.g. insect bite). The involved area is erythematous, edematous and pruritic. If only a local skin reaction (as opposed to generalized urticaria) is present, systemic manifestations (e.g. respiratory distress) are less likely. Local reactions, even if severe, are not predictive of systemic anaphylaxis on re-exposure.

Angioedema (soft-tissue swelling) is also commonly observed. These lesions involve the deeper dermal layers of skin. It is usually nonpruritic and nonpitting. Common areas of involvement are the larynx, lips, eyelids, hands, feet and genitalia.

Generalized (whole-body) erythema (or flushing) without urticaria or angioedema is also occasionally observed.

Cutaneous findings may be delayed or absent in rapidly progressive anaphylaxis.

Gastrointestinal Findings

Vomiting, diarrhea and abdominal distension are frequently observed.

DIAGNOSTIC STUDIES

Anaphylaxis is a clinical diagnosis in most cases.

LABORATORY STUDIES

Histamine and Tryptase Assessment

Plasma histamine levels rise within 10 minutes of onset but fall again within 30 minutes. Urinary histamine levels are generally not dependable, as this test can be affected by diet and by bacteria in the urine. Urinary histamine metabolites measurement is a better test but is not generally available.

Serum mature tryptase (previously called beta-tryptase) levels peak 60–90 minutes after the start of an episode and may persist for as long as 5 hours.

Basal levels of total and mature tryptase between episodes of anaphylaxis can be helpful to rule out systemic mastocytosis. Patients with mastocytosis constitutively produce large quantities of alpha-tryptase, while individuals with anaphylaxis from other causes have normal levels of alpha-tryptase at baseline between episodes of anaphylaxis. During anaphylaxis, a ratio of total tryptase (alpha and mature) to mature tryptase of 20 or greater is consistent with mastocytosis, whereas a ratio of 10 or less suggests anaphylaxis of another etiology.

Detecting the rise of histamine or tryptase levels can be difficult, and some patients might have a rise in one but not the other.

5-Hydroxyindoleacetic Acid Levels

If carcinoid syndrome is considered, urinary 5-hydroxyindoleacetic acid levels should be measured.

Serological and Skin Tests

Skin testing, in vitro IgE tests, or both may be used to determine the stimulus causing the anaphylactic reaction (e.g. food allergy, medication allergy, or insect bite or sting).

These tests cannot be used for non-IgE-mediated reactions.

INITIAL EMERGENCY DEPARTMENT INTERVENTIONS

The 2010 Joint Task Force Anaphylaxis Parameter Update, the 2011 World Allergy Organization anaphylaxis guidelines and the 2010 National Institute of Allergy and Infectious Diseases (NIAID)-sponsored expert panel report have similar recommendations for immediate treatment in the ED. It should begin with monitoring and treatment, including oxygen, cardiac monitoring, breathing, mental status, skin and a large-bore IV with isotonic crystalloid solution. At the same time, where appropriate, the ED team should call for specialized help, particularly a resuscitation team. Further intervention depends on severity of reaction and affected organ system(s), but the guidelines recommend the injection of epinephrine and placing the patient in a supine position (or position of comfort if dyspneic or vomiting) with the legs elevated.

Airway Management

For the initial assessment, check the airway closely. If needed, establish and maintain an airway and/or provide ventilatory assistance. Assess the level of consciousness and obtain blood pressure, pulse, and oximetry values. Place the patient in the supine position with legs elevated and begin supplemental oxygen.

One of the quickest and most effective ways to support ventilation involves a one-way valve facemask with oxygen inlet port [e.g. Pocket-Mask (Laerdal Medical Corporation, Gatesville, Texas) or similar device].

Artificial ventilation via the mouth-to-mask technique with oxygen attached to the inlet port has provided oxygen saturations comparable to endotracheal intubation. Patients with adequate spontaneous respirations may breathe through the mask.

Severe laryngeal edema may occur too rapidly during anaphylaxis and endotracheal intubation may be impossible. Epinephrine may rapidly reverse airway compromise. If the edema does not reverse with epinephrine, an endotracheal tube should be inserted.

In extreme circumstances, cricothyrotomy or catheter jet ventilation may be lifesaving when orotracheal intubation or bag/valve/mask ventilation is not effective. Cricothyrotomy is much easier that tracheostomy and is recommended if no surgical staff is immediately available.

Wheezing or stridor indicates bronchospasm or mucosal edema. Treatment with epinephrine and inhaled beta-agonists is effective for these indications. Inhaled beta-agonists are used to counteract bronchospasm and should be administered to patients who are wheezing.

Corticosteroids can be used but they do not have effect until several hours after the administration. Aminophylline can be more effective than corticosteroids in refractory bronchospasm.

For bradykinin-mediated angioedema (including angioedema due to ACE inhibitors), antihistamines and corticosteroids are probably not effective. Epinephrine may be tried in severe cases, but airway intervention may be needed.

Cardiac Monitoring

Cardiac monitoring is indicated due to the epinephrine and steroid use.

Intravenous Access

The IV line should be of large caliber due to the potential requirement for large-volume IV fluid resuscitation. Isotonic crystalloid solutions (i.e. normal saline, Ringer's lactate) are preferred. A keep-vein-open (KVO) rate is appropriate for patients with stable vital signs and only cutaneous manifestations. If hypotension or tachycardia is present, administer a fluid bolus of 20 mg/kg for children and 1 L for adults. Further fluid therapy depends on patient's response.

Epinephrine Administration

Epinephrine should be rapidly administered as a subcutaneous (SC) or intramuscular (IM) injection at a dose of 0.01 mL/kg of aqueous epinephrine 1:1,000 (maximum adult dose, 0.3–0.5 mL). The dose may be repeated q 5–10 minutes if there is persistence or recurrence of symptoms. Endotracheal epinephrine should be considered if IV access is not possible during life-threatening reactions.

Histamine Administration

Administration of H1 and H2 receptor antagonists is also recommended in the initial treatment of anaphylaxis.

- Administer diphenhydramine 25–50 mg IV or IM.
- Cimetidine 300 mg IV over 3–5 minutes, or ranitidine 50 mg IV, should be given initially; subsequent doses of H1 and H2 blockers can be given orally q 6 hours for 48 hours.

Corticosteroids

Corticosteroids are not useful in the acute episode because of their slow onset of action; however, they should be administered in most cases to prevent prolonged or recurrent anaphylaxis. Commonly used agents are hydrocortisone sodium succinate 250–500 mg IV q 4–6 hours in adults (4–8 mg/kg for children) or methylprednisolone 40–250 mg IV in adults (1–2 mg/kg in children).

Beta Agonists

Aerosolized β-agonists [e.g. albuterol, 2.5 mg, as-needed (PRN) every 20 minutes] are useful to control bronchospasm.

Atropine and Dobutamine

Additional useful agents in specific circumstances: Atropine for refractory bradycardia, dopamine for refractory hypotension (despite volume expansion), and glucagon in patients on beta-blocking drugs.

DISCUSSION

It is important to remember that anaphylaxis can be caused by often unpredictable causes. There have been instances where even ketchup has resulted in life-threatening anaphylactic shock. The author has personally seen medical staff crash within moments of eating French-fries and Ketchup.

Food Allergy Scenarios

1. A medical assistant went to a fast-food restaurant and had an anaphylactic reaction to a component in her ketchup. As her condition worsened she stopped breathing and was transported to the hospital via helicopter.
2. A young girl orders Chinese food at a sleepover, unaware that it contains peanuts. She experiences a fatal anaphylactic reaction.
3. A nurse, with a history of asthma, was intubated several times in an intensive care unit (ICU) following exposure to a perfume.

REFERENCES

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2. The Journal of Allergy & Clinical Immunology 2013 http://www.jacionline.org/

Case Study 55: Electrical Injury

CASE HISTORY

A 70-year-old male and his 25-year-old son were working on an irrigation system for their farm. When the son tried to attach the system to electricity he was shocked, his system was assaulted by 220 volts and immediately went into cardiac arrest (Fig. 1). The father brought his son, the sole breadwinner for the family, into the emergency department in full cardiac arrest and he was pronounced dead.

DISCUSSION

Electric current exists in two forms: Alternating current (AC) and direct current (DC). AC involves electrons flowing back and forth whereas in

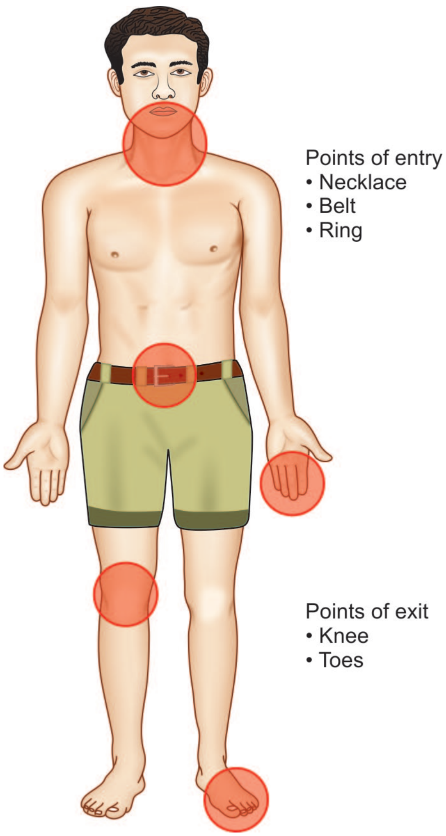


Fig. 1: A 25-year-old man assaulted by 220 volts

DC, the electrons flow in one direction only. AC is more dangerous than DC, because AC causes tetanic muscle contractions and the "locking on" phenomenon. The depth of electrical injury is related to the intensity and magnitude of the electric current. Ohm's law states that current = voltage/ resistance (Fig. 2).

Ohm's Law

$$\text{Current (amps)} = \frac{\text{Voltage}}{\text{Resistance}}$$

Since muscles, mucous membranes, blood vessels, and nerves have low resistance. They are the preferred pathway of electrical current.

Fig. 2: Ohm's law

Fig. 3: Electrical injury caused by chewing or biting electrical cords

Fig. 4: Flesh burns from electrical injury

PATHOPHYSIOLOGY

Direct necrosis of the myocardium from the electrical injury causes vasoconstriction, and ischemia which in turn will release excessive catecholamine leading to cardiac dysrhythmias. Even a small current can produce cardiac dysrhythmia like asystole and ventricular tachycardia. Burns are very common after high voltage electrical injury. Flesh burns from electrical injuries are flame burns because they are caused by ignition of clothing from electrical heat. In children, the most common form of electrical injury is caused by chewing or biting electrical cords (Figs 3 and 4). These cases present as perioral edema and eschar formation; the bleeding from perioral burns can be significant. Thorough physical examination is necessary to look for any entry and exit wounds. Immediate intravenous fluids are needed to establish fluid balance in all burn patients. Fluid resuscitation should be titrated to adequate urine output. Some patients may need admission to specialized burn units. Burns on both hands indicate electrical injury path going through the heart; this has a very poor prognosis.

Case Study 56: Anaphylaxis Reaction from Ace Inhibitor

CASE HISTORY

A 56-year-old woman is presented to the emergency department with severe edema of the lips, tongue and difficulty in breathing. Symptoms started suddenly after dinner. Patient's temperature is 100°F and she has elevated blood pressure (BP). She is tachycardic, tachypneic and her O_2 saturation level is 90% on room air. The patient is treated with oxygen, epinephrine 0.5 mg subcutaneously and Solu-Medrol 125 mg intravenously. Patient started feeling better in a few minutes.

Patient has no history of allergy to food, medication or environment. There is no previous history of atopic eczema or asthma. Past medical history has significant hypertension for which the patient is taking lisinopril 25 mg daily. Patient is treated in the emergency room and discharged with oral steroids, Zyrtec, and beta blocker and told to discontinue her medication. The next day the patient presented with the same symptoms; recurrent facial edema, and swelling of lips and tongue The treatment is repeated with epinephrine, Solu-Medrol 125 mg BID, oxygen and Benadryl 25 mg BID, and she is transferred to telemetry unit for stabilization and observation. She is discharged

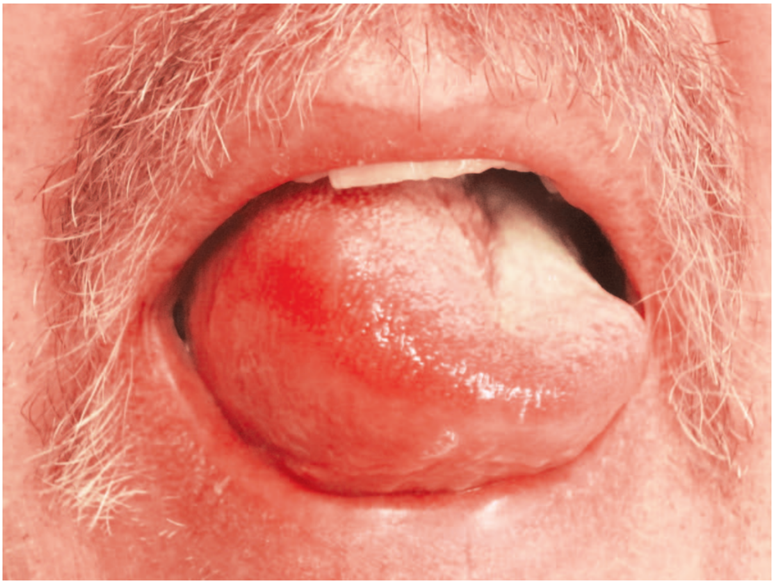


Fig. 1: ACE inhibitor-induced angioedema affecting half the tongue

Fig. 2: Angioedema affecting the tongue

3 days after treatment and diagnosed with angioneurotic edema secondary to angiotensin converting enzyme (ACE) inhibitor use. Patients with fulfillment allergic response, angioedema and airway compromise should be admitted and observed for more than 48 hours and closely monitored following discharge.

DISCUSSION

Angiotensin converting enzyme inhibitors are used for controlling BP, treating congestive heart failure, prevention of strokes, and prevention and progression of chronic kidney disease in diabetes or hypertension. ACE inhibitors are absolutely contraindicated in pregnant patients because they may cause birth defects. Patients with bilateral renal artery stenosis is also a contraindication for the use of ACE inhibitors. Cough and increase in potassium levels, dizziness and drowsiness are the main side effects for ACE inhibitors. The most serious and fatal side effects are kidney failure and serious allergic reaction (Figs 1 and 2). It can be anticipated, and early treatment intervention and withdrawal of the medication will prevent the morbidity and mortality secondary to ACE inhibitor use.

Case Study 57: Lightning Injury

CASE HISTORY

A 35-year-old male who was working on top of floor of the high-rise building during a thunderstorm came out to watch the thunderstorm. All of a sudden a lightning strike (Fig. 1) happened throwing the person to the ground. He was brought by the paramedics by ambulance to the emergency department. He is in cardiac arrest secondary to direct lightning strike. He was cardioverted. Immediate cardiopulmonary resuscitation (CPR) started and continued in spite of the fact that his pupils were fixed and dilated. Patient regained consciousness and started breathing spontaneously. There is no evidence of head and spinal cord injuries. He was bleeding from the left ear most likely secondary to rupture of the tympanic membrane (50% incidence). Electrocardiogram (ECG) and computed tomography (CT) head show negative results. Examination of the back in the thoracic area shows a fern-like skin pattern of the injury (Fig. 2). There is no evidence of compartment syndrome and neurological examination is normal. Intravenous (IV) fluids, normal saline are established for fluid resuscitation to have optimal urine output.

Fig. 1: Willis Tower in Chicago hit by lightning

Fig. 2: Lichtenberg figure (Pathognomonic of lightning)

DISCUSSION

Difference between lightning and electrical injury has been described in Table 1.

Table 1: Difference between lightning and electrical injury

	Electrical injury	*Lightning*
Type of current	*Alternating current*	*Direct current*
Occurrence	Common	Very rare
Fatality	Variable	25%
Direction of flow	Electrons flow back and forth the circle	Electrons flow in only one direction
Effects	More dangerous because it can cause tetanic contractions	Extremely high voltage and single intense muscle contraction that throws the victims causing fracture and spinal injury

Contd...

Contd...

Duration	*Prolonged*	*Brief*
Types of injury	"Locking on" phenomenon: Preventing the victims from the electrical source and prolonged exposure of the current	• Direct strike (most common and dangerous) • Side flash (other victims affected from the transmission of current from the first victim) • Ground current or strike potential • Flash over phenomenon
Complications		
• Renal	Renal failure secondary to rhabdomyolysis/myoglobinuria	Rare
• Cardiac	Dysrhythmia more common immediately after exposure	*Cardiac arrest*: Asystole
• Respiratory	Respiratory arrest rare	Respiratory arrest common secondary to injury to respiratory center in the medulla
• Cataracts	Rare	Common
• Tympanic membrane rupture	Rare	Common
• Skeletal injury	Fractures common	Fractures rare

MANAGEMENT/TREATMENT

Usually there will be multiple casualties at the time of lightning injury. Injuries typically take place on stormy days in outdoor locations or near high-rise buildings. Burns; linear, punctate, or fern-like pattern, are tell-tale clues. Rupture of the tympanic membrane with bleeding in the ear canal is also a very significant finding in the diagnosis of lightning strike. Immediate resuscitation as carried out on trauma patients is necessary. Airway, breathing, circulation, disability and exposure (ABCDE) should be followed by ECG, CT scan and tetanus, diphtheria, and pertussis (Tdap) vaccine are given if necessary. Visual acuity needs to be documented because cataracts are a common complication from this type of injury. A multidisciplinary approach; medical, nephrology, ENT, and ophthalmology for follow ups are necessary for the delayed effects.

Case Study 58: Bee Sting

CASE HISTORY

Chief Complaint

Patient cannot speak and comes in gasping for air.

History of Present Illness

A 50-year-old Latino male rushes into the emergency department with swollen neck and mouth. His throat is closing up, disabling him from breathing. Patient was eating lunch outside with sweetened lemonade. Shortly after lunch, patient realized a bee sting on his right hand dorsum.

Past Medical History

General health: good
Adult illness: Asthma controlled with inhaler, bB
Immunization history: Up-to-date
Screening history: Annual primary care physician (PCP) visits
Exercise: Sports
Tobacco: None
Alcohol: Glass of wine on weekends
Drugs: None
Medications: PRN bB inhaler for asthma attack
Allergies: Penicillin

REVIEW OF SYSTEMS

General: Fatigue, weak, fever. No weight change, chills or night sweats.
Skin: Itching, swelling and rashes on right hand dorsum. No skin, hair and nail changes. No sores, lumps or moles.
Eyes: Redness, tearing, itching of conjunctiva. Reading glasses. No contact lenses. No blurriness or acute visual loss.
Nose or Sinuses: Stuffiness. No rhinorrhea, sneezing, itching, allergy or epistaxis.
Mouth/Throat/Neck: No bleeding gums, hoarseness or sore throat. Laryngeal edema.
Cardiac: Dropping blood pressure. Peripheral edema. No murmurs, angina, palpitations, dyspnea on exertion, orthopnea or paroxysmal nocturnal dyspnea.

Respiratory: Shortness of breath, wheezing, dyspnea, asthma and cough. No sputum, hemoptysis, pneumonia, bronchitis, emphysema or tuberculosis.
Vascular: Peripheral edema. No claudication, varicose veins, thrombosis or emboli.

PHYSICAL EXAMINATION

General: 50-year-old Latino male in distress. Dressed in suit with clean hygiene.
Vitals: BP 90/70 mm Hg, pulse rate 120 beats/minute, respiratory rate difficulty in breathing, temperature 37.6°C.
Skin: Rash, swelling and redness.
Eyes: Pupils equal and reactive to light and accommodation, tearing, conjunctival injection. Anicteric sclera. No fundal papilledema. No hemorrhage. Lids normal. Extraocular movement normal. Visual fields and acuity normal.
Nose: Symmetrical. Nontender. Discharge present. Mucosa swollen. No inflammation of turbinate. Frontal and maxillary sinus nontender.
Mouth/Throat: Good hygiene. No dentures. No erythema, exudate or tonsillar enlargement. Laryngeal edema.
Lungs: Bronchoconstriction, cough, dyspnea, wheezing, asthma exacerbation. Chest symmetry with respirations. No crackles, vocal fremitus, whispered pectoriloquy and diaphragmatic excursion.
Vascular: Edema. Weak 1+ bilateral peripheral pulses. No bruit, jugular venous distention and varicose veins.
Lymphatic: No lymphadenopathy.
Neurologic: Within normal limits.

ASSESSMENT/PLAN

Impression

Type I hypersensitivity reaction: Anaphylaxis induced by a bee sting. Wheals and hives. Laryngeal edema. Hypotension. Tachycardia. Dyspnea. Gastrointestinal change.

Treatment

Urticaria: H1 antihistamines
Severe, acute urticaria: Diphenhydramine, hydroxyzine or cyproheptadine.

Life-threatening Anaphylaxis: Epinepherine, systemic Antihistamine and steroids (EPI Pen Fig. 3)
Anaphylaxis: Antihistamine with systemic steroids and epinephrine.
Chronic therapy: Non-sedating antihistamine, such as loratadine, desloratadine, fexofenadine and cetirizine.
Contraindication: No longer marketed; Astemizole and Terfenadine: potentially fatal rhythm disturbance when combined with macrolide antibiotics because of effect on hepatic P450.

DIFFERENTIAL DIAGNOSIS

- Asthma
- Syncope
- Panic attack

CONCLUSION

Type I hypersensitivity reaction mediated by IgE and mast cell activation resulting in wheals and hives.
Acute urticaria: Localized, cutaneous anaphylaxis. Hemodynamically stable without hypotension.

COMMON CAUSES

Allergic reaction: To medication, insect bites, foods and emotions.
Medication: Aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), morphine, codeine, penicillins, phenytoin, quinolones and angiotensin converting enzyme inhibitor (ACE-I).
Food: Peanuts, shellfish, tomatoes and strawberries.
Contact: Latex

Alert

Most deaths due to anaphylaxis occur within 30 minutes to 1 hour of insect bite or sting (Fig. 1). Airway management is a main priority in cases of anaphylaxis with angioedema (Fig. 2). Treating hypertension with recumbent position with legs elevated will help the victim.

PREVENTION

- Protective clothing
- Avoid common insect habitats and be aware of them, if possible
- Insect repellents which are not effective for bees and spiders

Fig. 1: European honey bee
Source: Accessed from wikipedia.org

Fig. 2: Periorbital swelling
Source: Accessed from wikipedia.org

Fig. 3: Pralidoxine auto-injector
Source: www.epipen.com

- *N,N-diethyl-m-toluamide (DEET)*: Most effective for mosquitoes, ticks, biting flies, fleas and chiggers
- Permethrin is an insect toxin if impregnated in clothing, will help prevent mosquito bites, tick bites, flies and chiggers
- Identify still present in skin, remove by flicking or scraping away from skin

Case Study 59: Lyme Disease

CASE HISTORY

A 22-year-old male who had been camping in Wisconsin Dells, Wisconsin is presented to the emergency room with a history of tick bites, complaints of feeling weak and tired, fever with chills, characteristic rash, generalized body ache, and headache for the past 3 days. He had a tick bite which he removed himself. He does not remember how long the tick was attached to his body. It seems to have been attached for more than 24 hours.

CASE DISCUSSION

The Centers for Disease Control and Prevention (CDC) defines erythema migrans as an expanding red macule or papule that must reach at least 5 cm in size with or without central clearing (Figs 2A and B). As for the Infectious Diseases Society of America (IDSA) guidelines, this rash is sufficient to make the diagnosis of Lyme disease in the absence of laboratory confirmation.

DIAGNOSTIC WORKUP

See Figure 1.

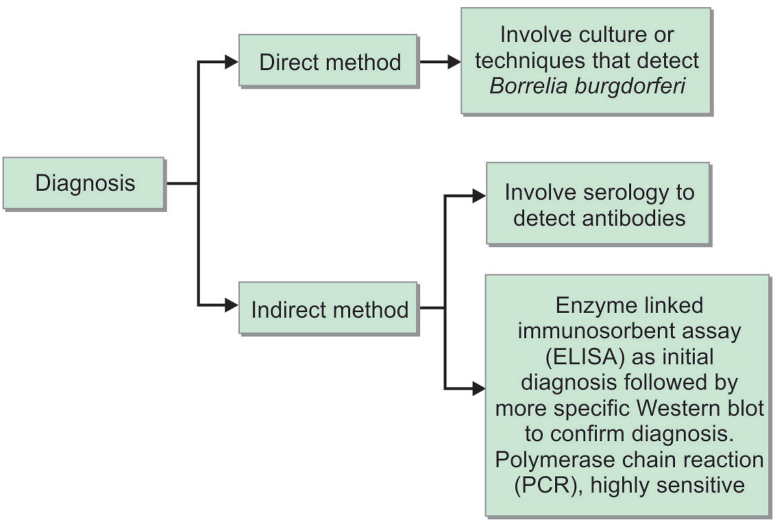
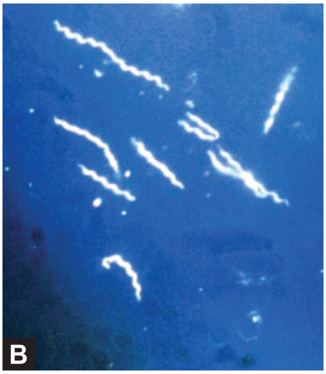


Fig. 1: Diagnostic Workup

TREATMENT

The treatment plan for Lyme disease is discussed in Flow chart 1



Figs 2A and B: Erythema migrans

Flow chart 1: Treatment plan

Treatment
- **Early localized**
  Doxycyline 100 mg PO BID or Amoxicillin 500 mg BID or Azithromycin 500 mg BID × 14–21 days
- Early disseminated → (Cardiac or neurologic complications) Ceftriaxone 2g IV or Cefotaxime 2g every 8 hours × 14-21 days
- Late
  - **Athritis** (without neurologic involvement. Oral antibiotics same as in early localized × 28 days
  - **Neurlogic** same IV antibiotics as given in early disseminated × 14–28 days

Case Study 60: Organophosphate Poisoning

CASE HISTORY

Patient is a 34-year-old farmer. One day he was working in his cornfield in Indiana spraying insecticide. After spraying his cornfield, he started to notice a tingling numbness, profuse sweating, salivation, lacrimation, coughing, palpitations, increased heart rate, increased urinary frequency and abdominal pain. He was in a confused state and having muscle cramps. He is brought into the emergency room after 30 minutes of this exposure. On arrival to the emergency room, he is decontaminated by giving a full bath to avoid further contact with the chemical. Stat (immediately) 2 mg of atropine is given under cardiac monitoring and repeated once more. Also, pralidoxime (2-PAM or 2-pyridine aldoxime methyl chloride) 1 g is given every 6 hours.

ORGANOPHOSPHATE POISONING

Mechanism

Organophosphates irreversibly bind to cholinesterase molecules. On the other hand, carbamates form a reversible bond with cholinesterase. Inhibition of cholinesterase enzymes leads to signs and symptoms of cholinergic crisis or excess. Exposure occurs most commonly through the dermal or oral routes. Frequently, hydrocarbons are present in the vehicle and if ingested, this must be considered as part of the toxic picture.

Clinical Effects

The clinical manifestations are mediated through two primary systems: muscarinic and nicotinic.

Muscarinic Effects

These include salivation, lacrimation, urination, gastrointestinal distress, bronchorrhea, bradycardia, bronchospasm, abdominal cramping and miosis.

Nicotinic Effects

These include altered mental status, hypertension, tachycardia and muscle cramps.

Laboratory Tests

Treatment should be guided by the signs and symptoms of toxicity. However, plasma and red blood cell (RBC) cholinesterase levels are rarely available at hospitals. If available, RBC cholinesterase levels give a better

reflection of what is happening at the nerve terminal. These values are generally only of value in retrospect. Fifty percent cholinesterase activity has mild toxicity. In a range of 10–20%, it shows moderate toxicity and if less than 10% shows severe toxicity.

Treatment

Decontamination is a critical part of managing dermal exposure. Atropine is given in 2 mg increment boluses until an adequate effect occurs. The most important endpoint is improvement of respiratory function and decreased secretions. The dose should be "enough" or until atropinisation occurs. Pralidoxime (2-PAM) 1 g is given every 6 hours in cases where atropine is required for treatment (Fig. 1). It works in organophosphate poisoning by dephosphorylating the cholinesterase enzyme permitting it to function. In carbamate poisoning, since the cholinesterase inhibition is reversible, 2-PAM is not necessary.

Center for Disease Control and Prevention Recommendations

Critical Care Area

If appropriate decontamination efforts have been completed before entry to the critical care area, there should be no need for special equipment or precautions such as covering floors and walls with plastic or shutting off the ventilation system. However, if the patient has ingested a chemical, then prepare to isolate toxic vomitus quickly (see "Ingestion Exposure" below).

Chemical burns have characteristics that are different from thermal burns. The extent and depth of injury in a chemical burn often is not apparent immediately; severity is frequently underestimated. Circulating fluid loss can occur as with thermal burns. In addition, absorption of a corrosive chemical may cause acute or delayed systemic toxicity.

Patients with exposure to a highly corrosive, penetrating, oily or persistent chemical may require additional decontamination to prevent further injury and systemic absorption. Common sites of residual contamination include the armpits, groin, buttocks, hair, ears, nostrils, and under the fingernails and toenails. Usually, these patients

Fig. 1: Pralidoxime chloride injection

do not pose a risk of secondary contamination if they have undergone the basic decontamination, but if the material is highly contaminating (e.g. organophosphate pesticides, radioactive dust), caregivers should wear gowns and gloves to protect themselves. Use plain liquid soap or shampoo for cleansing the skin.

Skin and Eye Contact

Skin and eye contact can occur with solids, liquids or gases. Corrosive agents can cause direct damage to tissues by various mechanisms including low or high pH, chemical reaction with surface tissue, or removal of normal skin fats (defatting) or moisture (desiccant effect). Chemicals can also be absorbed systemically through the skin. This is more likely to occur when the normal skin barrier is disrupted (e.g. with a chemical burn or a traumatic injury) or if the chemical is highly fat-soluble (e.g. organophosphate and organochlorine pesticides).

Central Nervous System

The brain is affected by many drugs and chemicals. Depressants (e.g. chloroform, hydrocarbon solvents) cause a generalized decrease in brain activity that may result in headache, dizziness, confusion, lethargy, stupor or coma. Some early effects of depressants may appear to be stimulatory, producing euphoria and giddiness (similar to beverage alcohol). Severe depression of the brainstem can cause respiratory arrest and cardiovascular collapse.

Central nervous system stimulants (e.g. DDT, other chlorinated hydrocarbon insecticides, organophosphates) can cause agitation, anxiety, delirium and seizures. Excessive muscular activity associated with seizures can result in hyperthermia.

Dermal

The skin provides a relatively impermeable protective barrier against excessive fluid losses from the body or inward movement of microorganisms, allergens and chemicals. Many chemicals disrupt the integrity of the skin by killing cells or removing fats from the skin. The barrier effect also may be lost by thermal burns or traumatic injuries. Disruption of the normal protective barrier can allow easier entry of chemicals into the systemic circulation. In addition, systemic illness can occur even without skin damage because many fat-soluble chemicals (e.g. some organophosphate insecticides) can penetrate intact skin.

Support Zone

As the support zone is set up away from the dangers of physical hazards or chemical exposure, contamination is not a serious problem in this area. Generally, personnel in the support zone does not require special protective clothing as long as victims have been decontaminated properly.

One important exception is exposure to a potent organophosphate pesticide or similar chemical; the support zone team should wear disposable aprons or gowns and latex gloves.

ADDITIONAL READING

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Case Study 61: Iron Poisoning in Children

BACKGROUND

Iron is a common accidental and intentional poisoning. It can carry a high morbidity and is one of the greatest causes of fatality in the pediatric age group from poisoning.

MECHANISM

Excessive iron is corrosive to the gastrointestinal (GI) system and can lead to hemorrhage. It can cause direct vasodilation leading to hypotension. It can affect most organs. It is a direct mitochondrial poison. Where it gets concentrated, disrupts oxidative phosphorylation and causes free radical formation leading to cell death. In the liver it causes "cloudy swelling" of the hepatocytes.

TOXIC DOSE

It is important to determine the elemental content of iron in each tablet (Tables 1 and 2).

STAGES OF IRON TOXICITY

1. *0–6 hours*: Nausea, vomiting, abdominal pain, diarrhea and hematemesis (in severe cases) can occur.
2. *Up to 12 hours*: A quiescent or danger phase occurs. One may develop a false sense of security in this phase.
3. *Starting at 6–12 hours*: This is the most serious phase including shock, GI hemorrhage, and hepatic toxicity.
4. *Weeks later*: This is the recovery phase where liver will be regenerated, if recovery has occurred. Gastric and intestinal stricture may be found at this time.

Table 1: Elemental iron content of various iron formulations

Formulation	*Elemental iron content*
Ferrous fumarate	33%
Ferrous sulfate	20%
Ferrous gluconate	12%

Table 2: Approach of treatment according to the dose of elemental iron uptake

Elemental iron dose	*Suggested management*
$<$ 20 mg/kg	Treat at home
$>$ 40 mg/kg (or lower if symptomatic)	Refer to hospital

LABORATORY TESTS

Iron levels should be obtained at 4 hours postingestion. Peak levels greater than 500 mcg/dL are considered toxic. Total iron binding capacity (TIBC) will be falsely elevated and is not necessary to be drawn. If the iron level exceeds the TIBC this confirms toxicity, but if not present does not exclude the toxicity.

Electrolytes may reveal an increased anion gap and metabolic acidosis.

White blood count and glucose levels greater than 15,000/mm^3 and 150 mg/dL respectively have previously been correlated with a toxic iron level. A recent study has shown these parameters to be unreliable; therefore, if these values are present they may suggest toxicity, but if absent, does not rule it out. An X-ray of kidneys, ureters and bladder (KUB) should be ordered to look for retained tablets. A positive KUB has been suggested as an indication for whole bowel irrigation with a polyethylene glycol electrolyte lavage solution (Golytely).

TREATMENT

Lavage should be done on all patients. There is no role for deferoxamine or phosphates in the lavage solution.

Deferoxamine therapy is optimally done as an intravenous infusion at 10–15 mg/kg/hr. A "vin-rose" color is a marker that iron is being removed from the body and excreted in the urine. However, the only important markers to follow are the persistence of an anion gap or low PH. Once this has resolved, deferoxamine can be discontinued. The deferoxamine should be used until the patient is free of systemic toxicity. It generally should not be used beyond 24 hours because of the risk of developing delayed acute respiratory distress syndrome (ARDS) from deferoxamine toxicity.

PREVENTION

An iron dose is the leading cause of fatalities in children less than 6 years of age. Fatal toxicity has been associated with more than 60 mg/kg of elemental iron ingestion. One tablet per kg is considered serious or fatal. Gastrointestinal decontamination with ipecac syrup or other mortalities is not recommended. It is crucially important to differentiate between a patient who is showing mild GI symptoms which shows resolving toxicity and a patient who is in latent phase of toxicity. The patient in latent phase needs to be observed, and treatment with deferoxamine is necessary.

Case Study 62: Carbon Monoxide Poisoning

CASE HISTORY

A 40-year-old male who was working in the garage for 2–3 hours on his car, without opening the garage because of cold weather, is brought to emergency department for severe headache, nausea and vomiting.

Carbon monoxide (CO) is an odorless, colorless gas that accounts for approximately 400 deaths annually. It is known as "the great imitator" and can typically cause vague symptoms mimicking other illnesses such as viral syndromes. CO is formed by incomplete combustion of carbonaceous fuels.

MECHANISM

There are four major mechanisms of toxicity:

1. Binding directly to hemoglobin causing a shift in the oxygen dissociation curve to the left
2. Direct cardiovascular depression
3. Inhibition of cytochrome
4. Lipid peroxidation (free radical generation)

CLINICAL EFFECTS

Symptoms impact all organ systems. Levels do not necessarily correlate with signs and symptoms; the guidelines are given in Table 1.

LABORATORY TESTS

Generally a level greater than 10% of carboxyhemoglobin confirms poisoning in smokers and a level higher than 5% confirms poisoning in a non smoker. Levels are as discussed above. Other important evaluations include creatine phosphokinase for the risk of rhabdomyolysis, electrocardiography for ischemia, neuropsychiatric testing and pulse oximetry or arterial blood gases (ABG). Findings of ABG include a normal PaO_2, a falsely elevated calculated oxygen saturation and a decreased measured oxygen saturation.

TREATMENT

Remove the patient from the source of exposure and administer high-flow 100% oxygen and immediate attention should focus on the airway breathing and circulation. Cardiac monitoring and IV line should be established.

Table 1: Signs and symptoms associated with carbon monoxide poisoning and correlated carboxyhemoglobin levels

COHb%	*Signs and symptoms*
0	None
10	Frontal headache
20	Throbbing headache
30	Impaired judgment, nausea, dizziness visual disturbance, fatigue
40	Confusion, syncope
50	Coma, seizures
60	Hypotension, respiratory failure
70	Death

Hyperbaric oxygen is indicated for more severe poisoning. Hyperbaric therapy is aimed at lowering the potential for delayed neuropsychiatric sequelae.

Indications for Hyperbaric Therapy:

1. History of coma or loss of consciousness*
2. Carboxyhemoglobin (COHb) greater than 25
3. COHb greater than 40, if transfer to chamber is required
4. Symptoms persisting more than 4 hours inspite of Oxygen therapy
5. Abnormal neuropsychiatric testing
6. Neonates
7. Pregnancy with a level greater than 10*
8. Low pH less than 7.2

PREVENTION

You Can Prevent Carbon Monoxide Exposure:

- Have your heating system, water heater and any other gas, oil, or coal burning appliances serviced by a qualified technician every year.
- Install a battery-operated or battery back-up CO detector in your home and check or replace the battery when you change the time on your clocks each spring and fall. If the detector sounds leave your home immediately and call 911(US) or 100 (India).
- Seek prompt medical attention if you suspect CO poisoning and are feeling dizzy, light-headed, or nauseous.
- Do not use a generator, charcoal grill, camp stove, or other gasoline or charcoal-burning device inside your home, basement, or garage or near a window.
- Do not run a car or truck inside a garage attached to your house, even if you leave the door open.

*Non-controversial indications

Fig. 1: Carbon monoxide alarm

- Do not burn anything in a stove or fireplace that isn't vented.
- Do not heat your house with a gas oven.

ADDITIONAL READING

1. http://www.cdc.gov/co/guidelines.htm

Case Study 63: Heat Emergency

CASE HISTORY

A 19-year-old athlete is presented to the emergency department for the chief complaint of very high fever, very dry and hot red skin, and tachycardic with feeble pulse with shallow breathing. He is confused and delirious. On arrival to the emergency room, his vitals are a temperature of 106.2°F, pulse rate 150 beats/minute, respiratory rate 22 breaths/minute, blood pressure 90/40 mm Hg, O_2 saturation 98% on room air.

DIAGNOSTIC WORKUP

Complete blood count (CBC) and comprehensive metabolic panel (CMP) is drawn and also urine analysis is performed.

Differential Diagnosis

See Table 1.

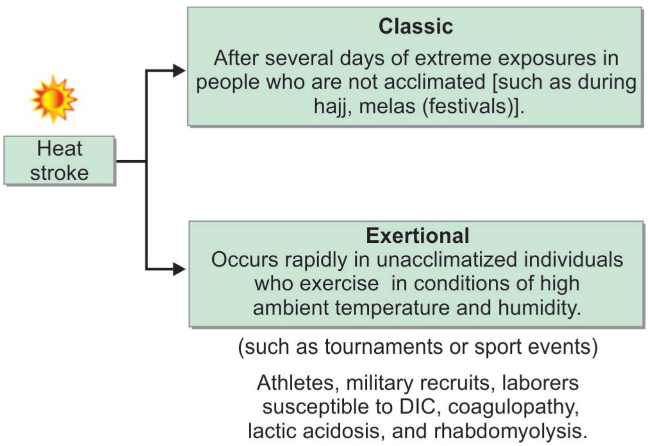
DIAGNOSIS

Heat stroke (Flow chart 1).

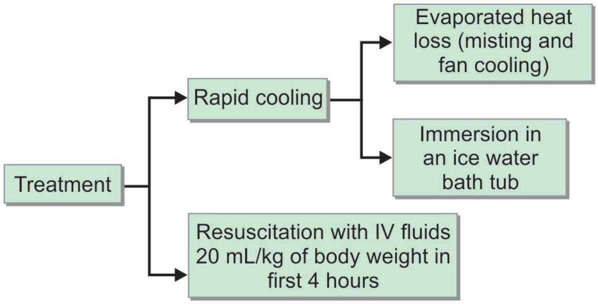
TREATMENT (FLOW CHART 2)

Intravenous (IV) fluid resuscitation, move victim to a cooler room, remove clothing, attempt a cold bath (sponging or wet sheet to reduce body temperature), watch for breathing problems, use fans and air conditioners, apply ice packs, deal with extreme caution.

Flow chart 1: Types of heat stroke



Flow chart 2: Treatment plan



Note: Stop cooling when temperature is 102°F (38.9°C)

Table 1: Heat-related injuries

Sunburn	Red skin, pain, possible swelling, blisters, fever, headaches	Remove oils that may be blocking pores by taking a shower. Be sure to not allow the body to cool naturally. For any blisters that occur, apply a dry, sterile dressing and seek medical attention.
Heat cramps	Pain normally in leg and abdominal muscles, profuse sweating	Take victim to cooler environment, ease muscle spasms by lightly stretching and massaging gently, every 15 minutes give half a glass of cool water (liquid should not contain caffeine or alcohol). If victim is nauseated, do not give any more liquids.
Heat exhaustion	Skin may be cool, pale or flushed but profuse sweating will occur, faint pulse, temperature likely to rise. Possible headaches, exhaustion, vomiting, nausea, dizziness or fainting.	Victim must lie down in a cool area. Remove clothing. Apply cool, wet clothes. Use a fan or air conditioner. If victim is conscious, give water. Allow victim to consume water slowly every 15 minutes. Do not give water if victim is nauseated. If there is vomiting, seek medical attention.

First Line Treatment

In heat exhaustion, hydration is necessary but no medication is required in initial management.

Second Line Treatment

Immunomodulators like corticosteroids can be used if necessary. Iced gastric, bladder, or peritoneal lavage may be necessary. Tantrolian 2–4 mg/kg of body weight is used to assist cooling.

Additional Treatment

Replacement of fluid and electrolytes with hypotonic oral fluids or IV, 0.5–1 L normal saline is done. Central venous pressure (CVP) monitoring may be necessary in extreme heat exhaustion and heat stroke. Body immersion in ice water and cooling the skin by evaporation by spraying water over the patient with convection by using fans is done. Immersing the hands and forearms in cold water is necessary.

Case Study 64: Amphetamines Overdose

CASE HISTORY

An 18-year-old student of 12th grade is brought to the emergency department by his parents early one morning for "behaving oddly". He reportedly stayed awake all night studying for one of his final examinations. His mother found him early in the morning making spaghetti for himself in the kitchen. Her son was extremely anxious and could not sit still or calm himself. At presentation, the boy is very talkative but emotionally labile. His temperature is 38.3°C, heart rate 120 beats/minute, respiratory rate 20 breaths/minute and blood pressure 140/90 mm Hg. His skin is flushed, and his pupils are dilated. Mild hyperreflexia is noted on physical examination.

DIFFERENTIAL DIAGNOSIS

- Substance abuse or amphetamines abuse
- Sepsis
- Encephalitis
- Meningitis or brain abscess
- Neuroleptic malignant syndrome (NMS)
- Malignant hyperthermia.

DISCUSSION

- Hyperthermia has a broad differential, and drugs of abuse should be kept in mind.
- Watch for rhabdomyolysis, disseminated intravascular coagulation (DIC) and multiorgan failure after hyperpyrexia.
- Amphetamines result in dopamine, norepinephrine and serotonin release, and catecholamine surge.

SYMPTOMS

- Patient may present with altered mental status, agitation, seizures, palpitations, chest pain, nausea, vomiting and diarrhea (Fig. 1).
- Severe hyperpyrexia
- Hyponatremia

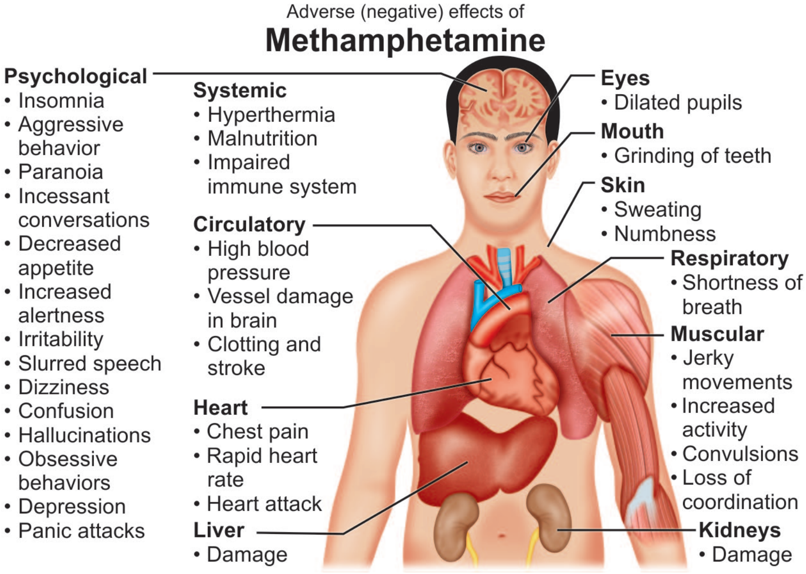


Fig. 1: Adverse effects of amphetamine overdose

COMPLICATIONS

- Rhabdomyolysis
- DIC
- Renal failure
- Hepatic necrosis
- Gastrointestinal bleeding
- Diarrhea
- Risk of vasculitics
- Neuropsychiatric abnormalities
- Damage to dopaminergic and serotonergic neurons
- Cardiomyopathy

Case Study 65: Marijuana (Cannabis) Abuse

CASE HISTORY

A 16-year-old girl is caught smoking marijuana with friends by her parents. She is now in the emergency department for assessment. She admits to smoking marijuana with these same friends on two other occasions over the past 6 months. She has also been to two parties where alcohol was available, and she states that she drank fewer than two beers at each event. She denies tobacco use and use of any other illicit drugs. She states that she is sorry that she "broke her parent's trust" and says she is motivated to remain abstinent.

DISCUSSION

Marijuana is a prevalent substance of abuse among teenagers and adults.[1] Two out of every five Americans admit to having tried marijuana in their lifetime, and one in ten have used in the past year. Marijuana is made with mixture of dried parts of the Cannabis sativa plant, and is usually smoked. Street names include pot, weed, bud, herb, ganja, hashish, grass and bhang (Fig. 1). Marijuana smoked as cigarettes are referred to as joints whereas pipes used to smoke it are called bongs. When cigars are used, they are referred to as blunts.[2]

Marijuana is the most commonly used illegal drug with a higher admission rate to treatment programs than all other drugs combined. Although marijuana is typically thought to be a milder drug, it can have systemic, multiorgan effects, and it can have a profound impact on all areas of one's life including their personal life and employment. The active ingredient in marijuana is delta-9-tetrahydrocannabinol (THC). Marijuana today has three times as much THC as that sold 20 years ago, making it much more potent. Puff for puff, smoking marijuana is more dangerous than smoking cigarettes, and its use has been linked to head and neck cancer.

Users often feel relaxed, have feelings of euphoria, increased heart rate, poor balance and coordination, slow reaction time, disorientation and panic. After the effects begin to fade, users begin to feel sleepiness, depression, and distrust or paranoia. Long-term marijuana use can impair learning and memory, and lower grades and poor work performance can result when people use marijuana.[3]

Fig. 1: Adverse effects of amphetamine overdose

Legally, marijuana has been used to treat several medical conditions including glaucoma, nausea that occurs with AIDS and cancer treatments, and the pain caused by multiple sclerosis. Marinol is the prescription form which contains marijuana's active ingredient of THC. Marinol is mainly used to treat nausea and vomiting.

The best method for marijuana abuse is prevention. Abstinence greatly increases the chance for a successful recovery. Patients may benefit from seeing a psychiatrist or psychologist for cognitive behavioral therapy to control their addiction.

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Case Study 66: Heroin Abuse

CASE HISTORY

An 18-year-old female is presented to the emergency department (ED) unresponsive via emergency medical services (EMS). According to EMS, her parents called 911 after finding her unresponsive in her bedroom. They say she had recently been exhibiting strange behavior at home, but they did not know she had been using drugs until they found her unresponsive with needles lying next to her on the floor. They could barely tell if she was breathing, but were able to find a faint pulse. En route, EMS started her on nasal cannula, placed two large bore intravenous (IV) lines, and administered Narcan subcutaneously. They noted that her breathing improved somewhat, but that she was still unresponsive. Vitals are temperature of 37°C, blood pressure 100/70 mm Hg, pulse rate 60 beats/minute, respiratory rate 8 breaths/minute, and O_2 saturation level 85% on nasal cannula. Physical examination reveals an unconscious female with unreactive pinpoint pupils and several track marks on her forearms bilaterally. The airway cart is prepared while the patient is administered 2 mg of naloxone intravenously. Patient's respiratory rate improves and she starts to regain consciousness. Two minutes later, another dose of 2 mg naloxone is administered. Now, the patient is awake and alert, sitting up in bed and vomiting. Laboratory tests, urine analysis and urine drug screening are ordered, and the patient is admitted for further monitoring of withdrawal effects.

DISCUSSION

Heroin is an illegal, highly addictive street drug.[1] It is commonly taken intravenously, but may be administered in other forms also (see Figs 1 and 2). Street names include junk, smack and skag. It is made from morphine which occurs naturally in the seed pods of poppy plants. Patients experience a sense of euphoria when using this drug. Over time, patients develop a tolerance and require more and more of the substance to achieve the same effect.

Symptoms of overdose include lack of breathing, shallow breathing or slow and difficult breathing.[2] Patients may also have dry mouth, pinpoint pupils, tongue discoloration, low blood pressure, weak pulses, bluish colored nails and lips, constipation, intestinal spasms, coma, delirium, disorientation, drowsiness and muscle spasticity. Skin examination may reveal track marks, or marks from "popping" heroin on the skin.

Fig. 1: Preparing Heroin for Injection
Source: www.wikipedia.com

Fig. 2: Heroin comes in brown or white powder
Source: Drug Enforcement Agency

Overdoses may be made more severe when the heroin is mixed with other drugs.[3] This may make treatment and recovery much more difficult.

Treatment should include airway support, fluids, laxatives and naloxone. Naloxone is an opioid antagonist which can reverse the effects

of the opioid. This can be very useful in cases where breathing or mental status is altered. However, Narcan may also precipitate severe withdrawal effects. Effects should be seen from naloxone within 5 minutes. Naloxone may be given 0.4–2 mg IV every 2–3 minutes pro re nata or when necessary (PRN). It may also be given intramuscularly, subcutaneously or via endotracheal tube or started on continuous infusion. If symptoms occur, naloxone may be administered every 1–2 hours. However, if there is no response initially after 10 mg, then an alternative diagnosis should be considered.

Heroin is highly addictive. Early symptoms include agitation, anxiety, muscle aches, increased tearing, insomnia, rhinitis, sweating and yawning. Later symptoms include abdominal cramping, diarrhea, dilated pupils, "goose bumps", nausea and vomiting. Unlike the heroin overdose, these reactions are not life-threatening.[4] Treatment for withdrawal involves supportive care. Clonidine may be used to reduce symptoms of anxiety, agitation, myalgias, sweating, rhinitis and abdominal cramping. Buprenorphine is also a promising medication for treating withdrawal symptoms and in fact has been shown to shorten the length of detoxification. Due to the severe withdrawal effects, methadone is often used to control these symptoms. The dose of methadone is then slowly decreased over time. Methadone is an opioid agonist that does not cause the same high of heroin, but limits withdrawal because it acts at the same receptors. Methadone has a very long half life and may be taken only once a day in order to help patients live a more normal, symptom-free life.

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Case Study 67: Alkaline Burns

INTRODUCTION

The following is a case report showing comparison of acid and alkaline burns sustained by patients involved in industrial accidents.

Alkaline burns are often more damaging than acid burns due to the subsequent liquefaction necrosis that occurs at the site of injury. This involves denaturation of proteins and saponification of fats, which allows extensive tissue penetration. The patient in this case is presented to the emergency department (ED) with sodium hydroxide burns to approximately 18% of his body, encompassing mainly the right leg. The patient was treated with irrigation of the wound, intravenous (IV) fluids and pain relief before transfer to a burn unit.

The damage from hydrofluoric acid burns results from the corrosiveness of the free hydrogen ions and the tissue penetration and coagulation necrosis caused by the acid. It additionally causes systemic toxicities due to depletion of total body calcium and magnesium, which results in enzymatic and cellular dysfunction. Most deaths are due to cardiac arrhythmias caused by hypocalcemia, which subsequently causes hyperkalemia.

CASE REPORT 1

A 26-year-old male is presented to the ED after a chemical burn injury to the right lower extremity. He had been standing on top of a chemical tank at work when he slipped and his leg fell into the tank, which was filled with sodium hydroxide.

He has no past medical history and social history is significant only for chewing tobacco.

On initial presentation, he has a blood pressure (BP) of 132/76 mm Hg, pulse rate of 89 beats/minute, respiratory rate of 20 breaths/minute and temperature of 97.9°F. He is found to have second degree burns to approximately 18% of his body encompassing the right leg and right lower quadrant of the abdomen (Fig. 1). He has blistering throughout the right leg and an area of black eschar on the right calf.

The wound is immediately irrigated with sterile saline and he is started on IV normal saline at 200 mL/hour. Additionally, he is given 30 mg of Toradol IV for pain relief. Vital signs remain stable and he is transferred to a burn unit.

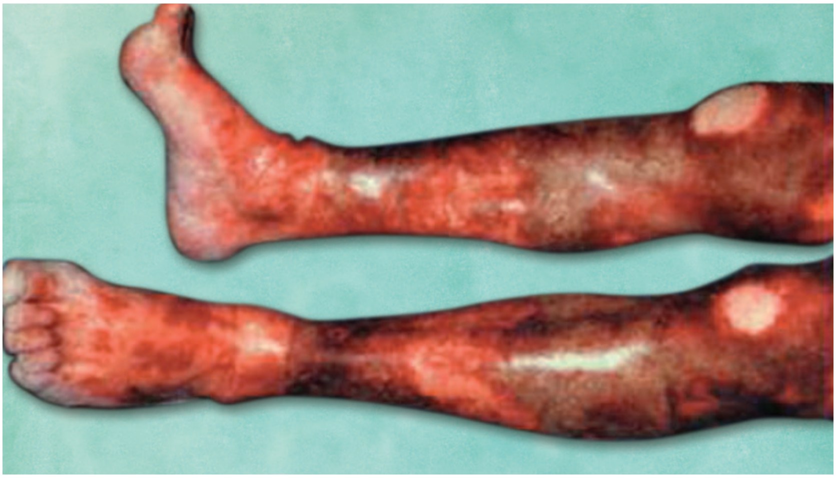

Fig. 1: Cement burns are one type of alkaline burn.
Source: Reprinted with permission from tunneltalk.com

CASE REPORT 2

A 29-year-old male is presented to the ED after a chemical burn injury to the right upper extremity. He had been working in a chemical plant when he dropped a container of hydrofluoric acid and sustained a splash injury.

He has no past medical history and social history is significant only for occasional alcohol use.

On initial presentation, he has a BP of 137/72 mm Hg, pulse rate of 91 beats/minute, respiratory rate of 20 breaths/minute and temperature of 98.8°F. He is found to have second degree burns to approximately 9% of his body encompassing the right arm. He has blistering throughout the right arm.

The wound is immediately irrigated with sterile saline and 2.5% calcium gluconate gel is applied. He is started on IV normal saline at 200 mL/hour and is given 30 mg of Toradol IV for pain relief. Vital signs remain stable and he is transferred to a burn unit.

DISCUSSION

The pathophysiology of chemical burns involves three zones of local response. The zone of coagulation is the point of maximum contact and is the site of irreversible damage due to coagulation of proteins. Surrounding this area is the zone of stasis, which is an area of decreased tissue perfusion. This area is potentially salvageable, if proper resuscitation is started quickly. Finally, there is zone of hyperemia in which there is increased perfusion. This zone will recover as long as prolonged hypoperfusion and sepsis do not develop.[1]

Sodium hydroxide is an extremely corrosive substance. Although it does not cause systemic toxicity, it causes severe burns to any area of body with which it comes in contact. It is especially damaging to the eyes due its ability to hydrolyze protein.

The standard of care of any chemical burn focuses on irrigation of the wound to decrease the length of exposure to the substance and fluid resuscitation to account for losses from the burned areas. There has been some research regarding whether neutralization with a weak acid would improve outcomes in alkaline burns. In rat models it has shown to result in increased depth of dermal retention, decreased leukocyte infiltration and improved epithelial regeneration when compared to those treated with only sterile saline irrigation.[2] More studies are needed to determine if there would be a benefit in humans as well. For hydrofluoric acid burns it is additionally important to apply calcium gluconate cream to the wound in order to neutralize the hydrofluoric acid.[3] It is also essential to monitor electrolytes and telemetry as arrhythmia due to hyperkalemia is a common cause of death.

As far as long-term treatments for all types of burns are concerned, there have been monumental advances in use of artificial skin for replacement after extensive burn injuries. The grafts are able to restore many of the physiological functions and anatomical structure of the skin, thus improving long-term outcome.[4]

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CHAPTER

10

Sexually Transmitted Infections (STI)

Case Study 68: Pelvic Inflammatory Disease

CASE HISTORY

A 23-year-old female is presented to the emergency department (ED) with left lower quadrant (LLQ) abdominal pain for 1 day. She reports a history of multiple sexual partners and was treated for chlamydia 1 year ago. Her only medication is oral contraceptive pills. On examination, her vital signs are temperature 101°F, pulse rate 87 beats/minute, respiratory rate 16 breaths/minute and blood pressure 118/78 mm Hg. On physical examination, she has moderate LLQ tenderness on palpation but no rigidity or guarding. She has cervical motion tenderness on bimanual examination.

Pelvic inflammatory disease (PID) is an infection of the reproductive tract (Fig. 1) and begins in the lower genital tract, and ascends into the endometrium, adnexa or peritoneal cavity. This can lead to salpingitis, endometritis, tubo-ovarian abscess (TOA), perihepatitis or focal pelvic peritonitis. It is almost always caused by *Neisseria gonorrhoeae* or *Chlamydia trachomatis* but 30–40% are polymicrobial. Risk factors for PID include multiple sexual partners, sexual abuse, adolescence, presence of

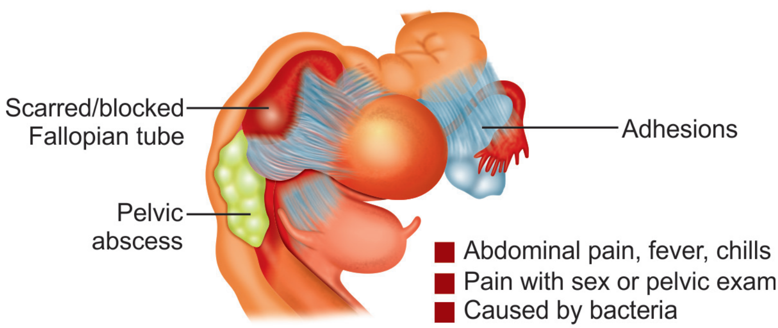


Fig. 1: Anatomy of female reproductive system

other sexually transmitted diseases, douching and intrauterine devices. It is uncommon in pregnancy but can lead to fetal loss, if it occurs in the first trimester. Long-term complications include ectopic pregnancy, infertility, and chronic pain.

CLINICAL PRESENTATION

The most common feature is lower abdominal pain. Other symptoms include vaginal discharge, vaginal bleeding, dyspareunia, urinary discomfort, fever, nausea and vomiting. Peritoneal signs may also be present. Patients presenting with right upper quadrant pain with jaundice should be suspected of having Fitz-Hugh-Curtis syndrome or perihepatitis.

DIAGNOSIS

Diagnosis begins with a pregnancy test, wet prep, and endocervical swabs for gonorrhea and chlamydia. Elevation of white blood cell count, erythrocyte sedimentation rate and C-reactive protein also suggest the diagnosis. A pelvic ultrasound can be used to detect TOA. It is important to remember that PID may mimic surgical conditions such as appendicitis, cholecystitis and ovarian torsion. Differential diagnosis also include: Diverticulitis, ectopic pregnancy, spontaneous or septic abortion, ovarian cyst, pyelonephritis and renal colic. Table 1 shows the diagnostic criteria for PID.

Table 1: Center for Disease Control and Prevention recommended diagnostic criteria for pelvic inflammatory disease

Table 1: CDC diagnostic criteria for PID
PID should be suspected and treatment is initiated if:
• Patient is at risk of PID
and
• Patient has uterine, adnexal or cervical motion tenderness with no other apparent cause
• Findings that support the diagnosis
• Cervical or vaginal mucopurulent (green or yellow) discharge
• Elevated erythrocyte sedimentation rate or C-reactive protein
• Laboratory confirmation of gonorrheal or chlamydial infection
• Oral temperature of 101°F (38.3°C) or greater
• White blood cells on vaginal secretion saline wet mount
• Most specific criteria for the diagnosis
• Endometritis on endometrial biopsy
• Laparoscopic abnormalities consistent with PID
• Thickened, fluid-filled tubes apparent on transvaginal ultrasound or magnetic resonance imaging

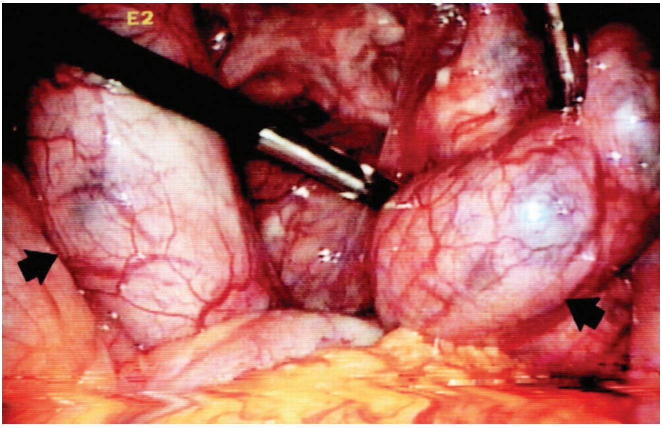


Fig. 2: Pyrosalpinx in pelvic inflammatory disease (PID)
Source: Reproduced with permission from RSNA.org

TREATMENT

Treatment should begin with adequate analgesia and hydration. The need for inpatient management should be based on toxic appearance, inability to tolerate oral medication, inability to exclude alternative surgical diagnoses, pregnancy, adolescence, immunosuppression, or suspected anaerobic infection due to intrauterine devices, suspected abscess or recent instrumentation. If TOA is diagnosed, 60–80% of patients will respond to antibiotics alone while the remainder will require drainage. Patients managed on an outpatient basis should follow-up within 72 hours, and the patient and sexual partner should complete the full course of treatment in order to prevent reinfection. Preventive counselling and HIV testing should also be provided.

The following are options for outpatient antibiotic therapy:

1. Ofloxacin 400 mg PO BID for 14 days or levofloxacin 500 mg PO QID for 14 days with/without metronidazole 500 mg PO BID for 14 days.
2. Ceftriaxone 250 mg intramuscularly (IM) once or cefoxitin 2 g IM once and probenecid 1 g PO × 1 + doxycycline 100 mg PO BID for 14 days with/without metronidazole 500 mg PO BID for 14 days.

If managed as an inpatient, the following treatment options are recommended.

1. Cefotetan 2 g intravenously (IV) q12 hours or cefoxitin 2 g IV q6 hours with doxycycline 100 mg IV or PO q12 hours.
2. Clindamycin 900 mg IV q8 hours with gentamicin 2 mg/kg IV loading dose followed by 1.5 mg/kg q8 hours.
3. Ofloxacin 400 mg IV q12 hours or levofloxacin 500 mg IV q24 hours, and doxycycline 100 mg PO or IV q12 hours with/without metronidazole 500 mg IV q8 hours or ampicillin-sulbactam 3 g IV q6 hours.

CHAPTER

11

Pediatrics

Case Study 69: Acute Appendicitis

CASE HISTORY

Patient is a 14-year-old male who is seen in the emergency room for abdominal pain. The pain started with a dull periumbilical pain radiating to the right lower quadrant (RLQ). He feels anorexic, nauseated and vomited twice since morning. Over the past 24 hours, he also complained of dysuria and colicky abdominal pain. Abdominal examination shows a positive McBurney's sign, Rovsing's sign, positive psoas sign and obturator sign. Rectal examination was painful. On examination, his temperature is 102.2°F. Abdominal examination shows some abdominal rigidity. Complete blood count (CBC) shows white blood cell count of 14,700/mcL3 with left shift, polymorphonuclear neutrophil (PMN) is 90%, urinalysis (UA) is positive for RBCs and leukocytes. Abdominal ultrasound and computed tomography (CT) of abdomen are both negative.

DIAGNOSIS

Acute appendicitis; most likely this is a pelvic appendicitis. A pelvic CT was ordered and diagnosis of acute appendicitis confirmed (See Figure 1A).

DISCUSSION

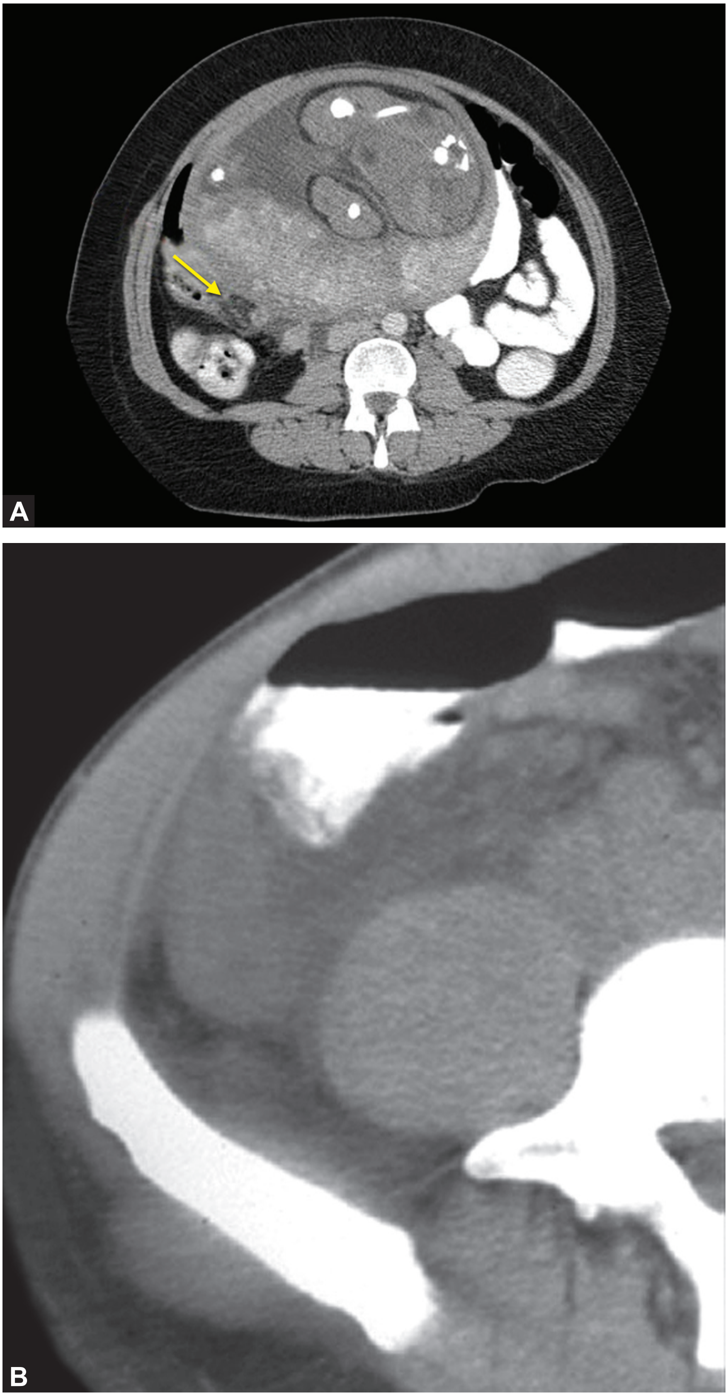
The incidence of appendicitis is approximately 6% of the general population. Nowadays, unnecessary appendectomies are avoided because of the new imaging techniques. However, there are still some cases in which the classic signs and symptoms are absent, still leading to continued difficulties in diagnosis. Abdominal pain is still the most reliable symptom in the appendicitis. Fever is a relatively late finding unless there are complications or it ruptures.

Appendicitis is the inflammation of the appendix. Obstruction by fecalith, foreign bodies or tumor promotes inflammation and infection, leading to swelling. The distended appendix then touches the peritoneum and irritates it, which presents as the classic acute abdomen typified by involuntary guarding and rebound tenderness. The treatment is appendectomy. If left untreated and the appendix continues to swell, its blood supply can be compromised. The appendix can then infarct and rupture, causing widespread peritonitis, high fever and septic shock. Therefore, acute appendicitis needs to be treated as a medical emergency.

The clinical symptoms of acute appendicitis may vary widely. The symptoms typically start with pain in the umbilicus or the epigastrium, for which the visceral pain of the inflamed appendix is referred. As the inflammation spreads throughout the intestinal wall, the visceral peritoneum irritation localizes the pain to the RLQ. McBurney's point, or one-third of the distance from right anterior superior iliac spine (ASIS) to the umbilicus, is the most cited location of pain associated with appendicitis in literature. Other symptoms such as nausea and vomiting may be present. Anorexia, on the other hand, is almost always present. Although less definitive, the presence of mild leukocytosis along with the presenting symptoms also points to acute appendicitis.

Several clinical examinations and studies are used in aid of diagnosing acute appendicitis. Clinical pain evaluation includes Rovsing's sign, psoas sign and obturator sign. In Rovsing's sign, deep palpation of the left lower quadrant (LLQ) pushes the visceral contents to the right and causes RLQ pain. Psoas sign is pain elicited by having the patient lying on the left side and either passively extending the hip or actively flexing the hip. This is due to inflammation of the overlying peritoneum and/or the psoas muscle, which may be in contact with the appendix. Obturator sign is documented with internal rotation and flexion of the hip joint. This maneuver puts the obturator externus muscle in contact with the enlarged appendix, thus causing pain. Although supportive, these three techniques to evaluate pain are not diagnostic of appendicitis.

Imaging studies provide better sensitivity and specificity of diagnosing acute appendicitis. Abdominal ultrasound can achieve a sensitivity of 90%, although the results are highly operator-dependent. CT scan with intravenous (IV) contrast has above 95% sensitivity and specificity (Figure 1A shows with contrast and 1B shows without contrast), although a negative result does not rule out the diagnosis. CT scan is now considered the imaging study of choice- not ultra sound. We have to be very careful in patients who are pregnant because nausea and vomiting may be incorrectly linked to pregnancy. We have to remember appendicitis is the most common extra-uterine surgical emergency in pregnancy. The fetal mortality rate will be very high if the appendix ruptures and peritonitis



Figs 1A and B: (A) CT abdomen (ABD) with contrast; (B) CT scan without intravenous (IV) contrast. The contrast shows appendiceal wall changes and periappendiceal fat changes

sets in. In these cases, ultrasound is the preferred diagnostic tool due to the risk of radiation to the newborn from CT scan. Patients with questionable diagnosis should be observed with serial abdominal examinations to avoid premature surgical interversion or discharge.

For this reason, approximately 20% of appendectomies result in normal appendices.

In atypical acute appendicitis, the patient may not present with all the symptoms of typical appendicitis. Clinicians must rely on the patient's history, imaging studies, and clinical experience to make the appropriate diagnosis.

Incidence of acute appendicitis peaks in teenage years to mid-twenties. Important differential diagnoses include ectopic pregnancy, ruptured ovarian cyst, Merkel's diverticulitis, intussusceptions, pelvic inflammatory disease and other disorders associated with abdominal pain.

Emergency Room Treatment

Patients need to be NPO (nothing by mouth). IV access, analgesia, and antibiotics.

- *Analgesia*: Fentanyl 1–2 micro-grams/ kg IV every 1–4 hours
- *Antibiotics*: Ampicillin/sulbactam 3 g IV or piperacillin/tazobactam 3.375 g IV
- Patient care can be divided into 4 subgroups:

Group 1: With classic appendicitis will go for surgical consult and appendectomy

Group 2: Patients suspicious for appendicitis will go for serial imaging and serial examinations.

Group 3: High risk patients like pediatric, geriatric, or pregnant will need surgical consult and follow up.

Group 4: Non-specific abdominal pain. Close follow up and specific discharge instructions to follow up with primary care provider or return to the emergency room if the symptoms return.

ADDITIONAL READING

1. Lennard Nadalo. Appendicitis: Avoiding Pitfalls in Diagnosis. Medscape.
2. Source: http://en.wikipedia.org/wiki/Appendicitis
3. Emergency Medicine: A Comprehensive Study Guide, 6th ed, see Chapter 78, "Acute Appendicitis," by Denis J. FitzGerald and Arthur M. Panciolli.

Case Study 70: Child Abuse

"If I prevent one child from being abused and one child from drowning, I'll consider myself fortunate."

—Badar M Zaheer

(Studies show Child Abuse and Neglect Cost the United States $124 Billion)

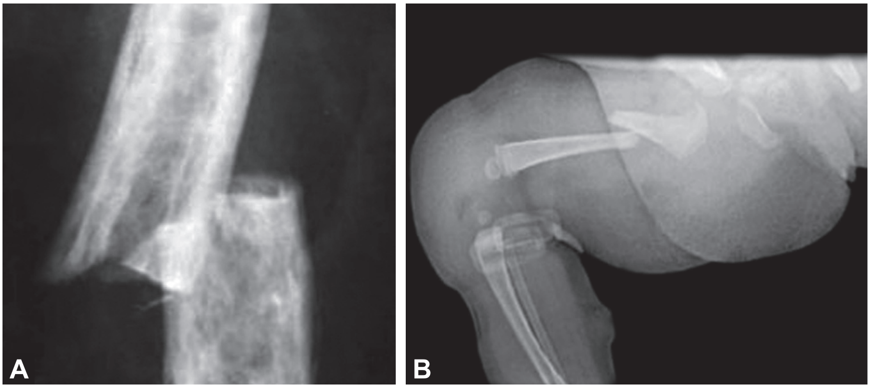
CASE HISTORY

The patient is a 6-year-old boy accompanied by his mother, who presents to the emergency room with complaints of right leg pain. Mother reported that the patient had fallen down a flight of stairs earlier in the day and has been unable to walk since then, secondary to pain. During the patient encounter, the child remained quiet and mother answered all questions which were asked. Mother asked several questions regarding what the plan was and what tests the doctors were planning on running. The patient was sent for an X-ray of the leg to evaluate for possible fractures.

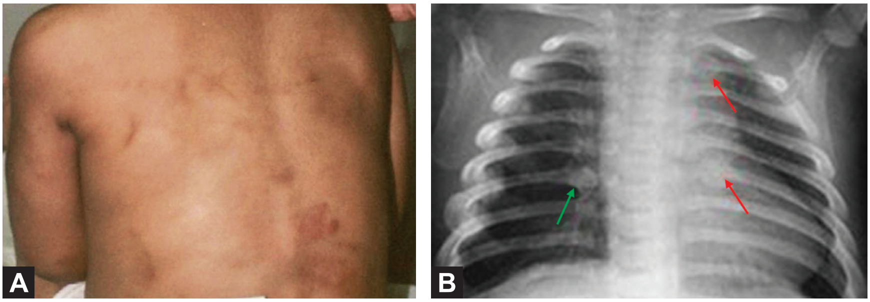
The results of the lower extremity X-ray demonstrated a transverse fracture of the tibia (Figs 1A and B). The radiologist also noticed some old healing fractures on the lower extremity and recommended a skeletal survey to check for other healing fractures. When the mother was informed that her son needed to get some more X-rays, she asked why they wanted to expose the child to unnecessary radiation. She denied that her son had suffered any previous trauma, but this poor child suffered a fracture to the base of skull and multiple rib fractures (Figs 2 and 3).

DISCUSSION

Current research states that 3% of all children seen in the emergency department for child abuse will return with a secondary maltreatment diagnosis within 1 year.[1] In the United States, there are 3.3 million cases



Figs 1A and B: Femur fracture of a 50-day-old infant who also suffered from head injuries



Figs 2A and B: A child suffering from a fracture to the base of skull and multiple rib fractures
Source: Wikipedia

Fig. 3: Circumferential fingerprint bruising on the arm of a 6-month-old child

of child abuse/neglect reported annually with an estimated cost of over $100 billion. Child abuse is not limited by socioeconomic level, culture, religion, or education level. The best way to prevent child abuse, according to the Centers for Disease Control and Prevention (CDC), is to stop it before it starts (Fig. 4). As health care professionals, we must promote the development of safe and stable relationships between children and their parents/caregivers to prevent abuse before it starts.

Unless our governments act to enforce laws to prevent child abuse, the work of doctors will not be enough.

Community education and involvement is essential to prevent child abuse. Free reporting without obligation and without a burden of proof on the witness should be made compulsory. Those who do not report cases should be punishable by law, especially medical professionals, governmental authorities, teachers, school administrators, etc. It is a team effort to prevent this, and a universal responsibility on the shoulders of all human beings.

Fig. 4: Reporting child abuse

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1. Osterweil N. Abused Children Treated in ED at Risk of Return. Annual Meeting of the Pediatric Academic Societies. Elsevier Global Medical News. May 15, 2012.

ADDITIONAL READING

1. Child Abuse and Neglect cost the United States $124 Billion, Study says.
2. www.cdc.gov/safechild.

Case Study 71: Intussusception

CASE HISTORY

A 5-year-old boy is presents to the emergency department with the chief complaint of abdominal pain colicky in nature associated with vomiting, has fever since morning and looks dehydrated. He is drawing his legs up to control the pain when he gets abdominal colic. Mother noticed that he is passing currant jelly stools (Fig. 1). Physical examination shows low skin turgor, decreased capillary refill and tongue slightly dehydrated. Examination of the abdomen shows diminished bowel sounds and palpable mass which looks like sausage. Anal examination shows some rectal prolapse like appearance. His vitals are pulse rate of 150 beats/minute, respiratory rate 28 breaths/minute and temperature 99.9°F. Diagnostic test includes abdominal X-ray obstructive series which show absence of gas in the right upper quadrant and ultrasonography shows telescopic bowel.

DIAGNOSTIC TEST

Diagnostic test includes abdominal X-ray obstructive series which show absence of gas in the right upper quadrant and ultrasonography shows telescopic bowel (Fig. 3). CT scans are also useful when indicated (Fig. 2).

Fig. 1: Information about this image: Currant Jelly Stool
Courtesy: of www.virtualpediatrichospital.org/

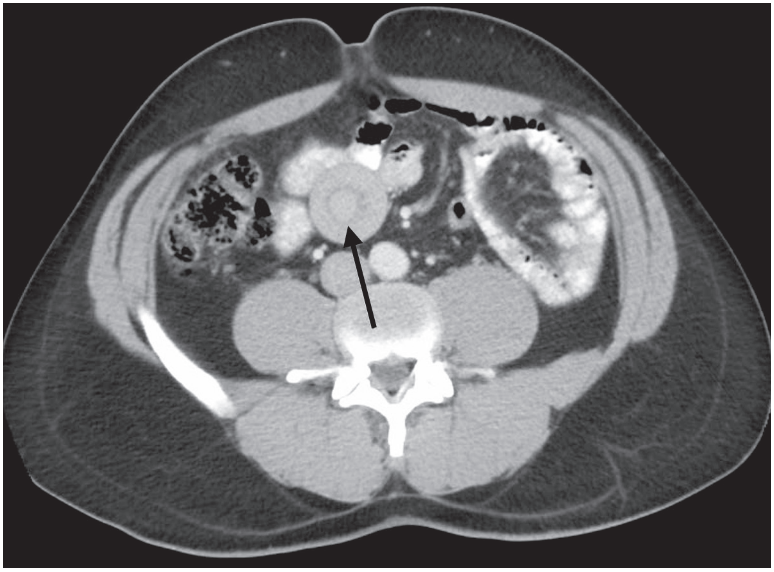


Fig. 2: CT Scan of intussuception
Courtesy: Wikipedia

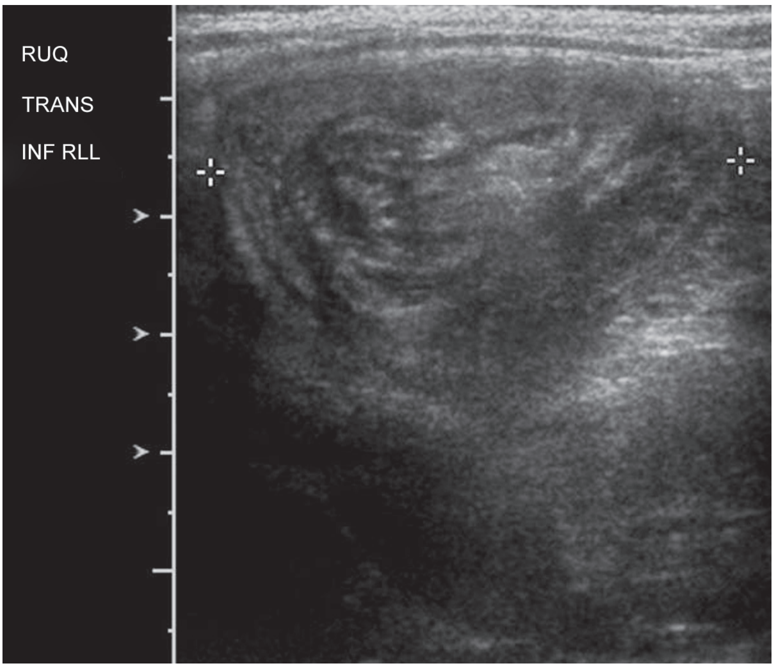


Fig. 3: Ultrasound image of Intussuception
Courtesy: Wikipedia

DISCUSSION

Intussusception is the most common cause of bowel obstruction in children from 3 months to 6 years of age. It occurs when a portion of the bowel "telescopes" into itself, causing intestinal obstruction. The intestinal obstruction may progress to a segment of the intestine. The condition can progress from intestinal obstruction to necrosis (tissue death) of a segment of the intestine. Initially, blood supply to the intestine is compromised causing edema and inflammation which leads to perforation, peritonitis, shock and death.

ETIOLOGY

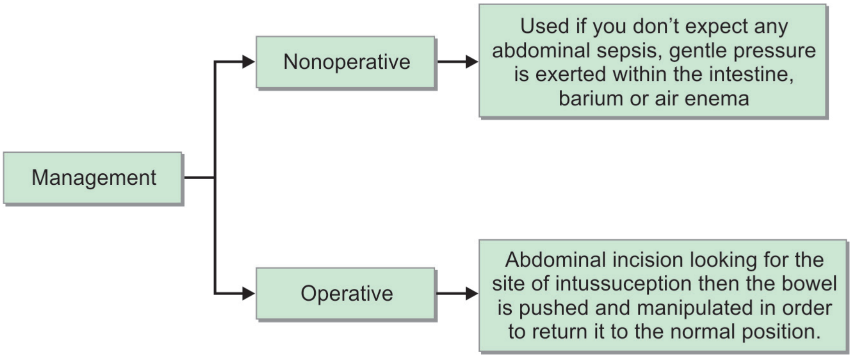
- Idiopathic
- Viral infection
- *Cystic fibrosis*: Patients suffering from this disease are more prone to get intussusception because of change in mucosa. Another underlying condition such as hematoma from trauma or following surgery.

TREATMENT

Management of intussusception through nonoperative and operative procedures is shown in Flow chart 1 and Fig. 4.

- *Intravenous fluid*: Measure intake and output of fluid
- Maintain NPO status as ordered
- Insert nasogastric tube, if ordered to decompress stomach.
- Continually reassess condition because increased pain and bloody stools may indicate perforation.
- After reduction by hydrostatic enema, monitor vital signs and general condition.

Flow chart 1: Management plan of intussusception



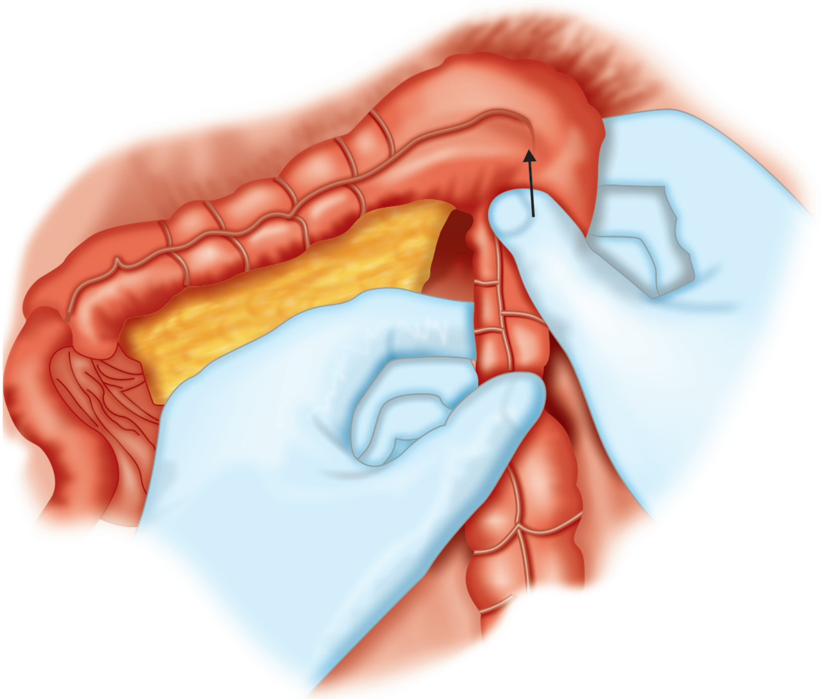


Fig. 4: Operative management of intussusception. Technique for reduction of an intussusception in the operating room. The intussusception is being squeezed by the surgeon rather than pulled[2]

Although intussusception is relatively common in pediatrics, significant variability in diagnosis and treatment modalities exist. The management of children after a successful enema reduction is an area that has not been well studied. Now it is well-documented that recurrences of intussusception can be reduced safely with enema techniques with a success rate of up to 95%, and that the incidence of pathologic lead point (PLP) is not increased until the patient has more than one recurrence.[1] There was an association between rotavirus and intussusception and some of the earlier vaccines had to be recalled. Recent vaccines are not supposed to have the same effect.

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2. Daneman A: Intussusception: Issues and controversies related to diagnosis and treatment. Radiol Clin North Am 34:743, 1999.

Case Study 72: Malrotation and Volvulus

CASE HISTORY

A 1-year-old boy with his parents is presented in the emergency department (ED) with abdominal pain. The pain started last night and is associated with bilious vomiting. Today he had a bloody bowel movement. Past medical and surgical history is insignificant. Immunization is up-to-date. Prenatal and postnatal history is unremarkable..

PHYSICAL EXAMINATION AND LABORATORY VALUES

On examination, mild to moderate distension and diffuse tenderness with guarding of the abdomen noticed.

Laboratory Tests

Complete blood count (CBC) and electrolytes are within normal limits (WNL).
Stool: Positive for occult blood. Radiography results are as shown (Figs 2A and B). Dilated loops of bowel overlying the liver shadow and little gas distal to the obstruction noted.

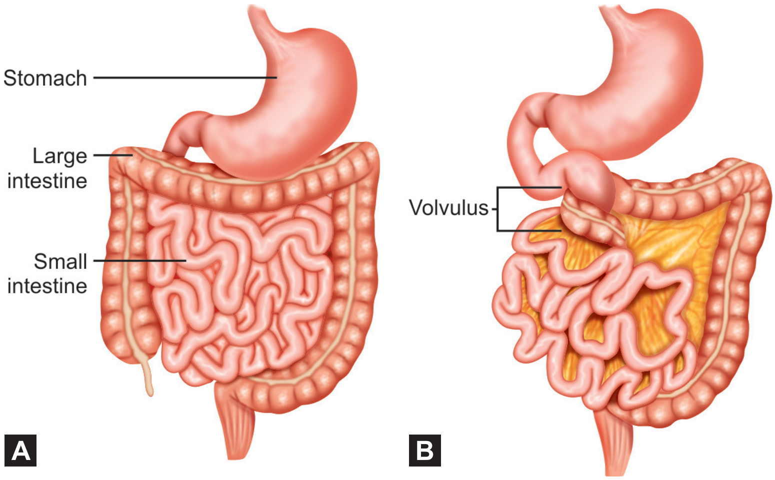
DISCUSSION

Malrotation of the gut occurs in approximately 1 in 500 births.[1] The gut rotates itself into the abdominal cavity during development. If this process does not proceed properly, the intestines may be out of their usual locations. The mesentery stalk, or the connective tissue that fixes the bowels to the abdominal wall and channels blood vessels and lymphatics, may not form properly. The mesentery is usually a broad band that secures its connection. In a malrotated gut, the mesentery may be thin, making the intestines more prone to twist on themselves. In individuals with gut malrotation, the most common form of twisting is mid-gut volvulus, in which the intestines twist around the superior mesentery artery (Figs 1A and B).

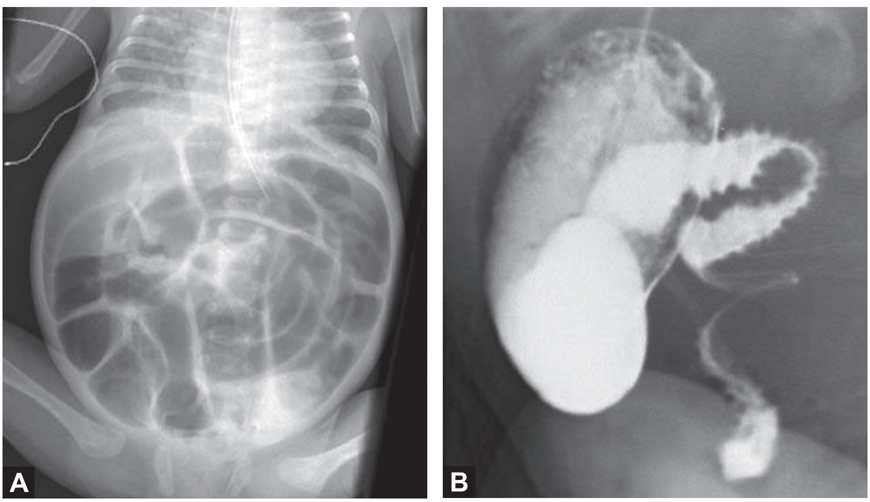
Mid-Gut Volvulus

Mid-gut volvulus is commonly seen in the pediatric population due to its congenital etiology. The patient presents with bilious vomiting, abdominal pain, lethargy, crying, and failure to pass stool or gas. Other symptoms such as hematemesis, rectal bleeding, or signs of sepsis suggest perforation or infarction of the bowels.

The gold standard for diagnosing volvulus is an upper gastrointestinal (GI) series, or barium swallow. With the help of oral contrast, abdominal X-ray may show the "coffee bean" sign, which is formed from two air bubbles proximal and distal to the volvulus (Fig. 2A). A barium enema may also be performed, which may show the "bird's beak" sign at the point of volvulus (Fig. 2B).[1] Differential diagnoses include carcinoma or other forms of intestinal obstruction.



Figs 1A and B: Mid-gut volvulus. The large intestine twists around the duodenum and superior mesenteric artery, which jeopardizes the entire mid-gut



Figs 2A and B: (A) The "coffee bean" sign in which two bubbles are formed around the volvulus; (B) Example of "bird's beak sign in barium enema study

TREATMENT

The treatment is emergency pediatric surgery. Ladd's procedure is usually performed. After the surgeon untwists the volvulus, he or she frees malformed connecting stalk and fixes the intestines to the abdominal wall. The small intestine is usually fixed to the right side of the abdominal cavity, while the large intestine fixed to the left.[1] Because this is not the normal positioning of the usual anatomy, prophylactic appendectomy is usually done to avoid diagnostic confusion if the patient later suffers from appendicitis. If the surgeon determines a segment of the bowels is necrotic, resection of that segment would be performed.

Many individuals with malrotation do not develop volvulus. In fact, many do not know about their condition until they have either imaging studies or surgeries unrelated to their malrotation.

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1. Ingoe R, Lange P. The Ladd's procedure for correction of intestinal malrotation with volvulus in children. AORN J. 2007;85(2):300-8.

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1. Duggan CP. (2011) Intestinal malrotation. [online] Available from www.childrenshospital.org/az/Site1181/mainpageS1181P0.html. [Accessed August, 2012].
2. Nemattalla W. (2010). Sigmoid volvulus. [online] Available from radiopaedia.org/cases/sigmoid-volvulus. [Accessed August, 2012]

Case Study 73: Acute Laryngotracheitis/Croup

CASE HISTORY

A 2-year-old child is brought by her parents for the chief complaint of harsh barking cough, hoarseness of voice and a mild stridor. The child had upper respiratory tract infection symptoms for the past 3 days with low rate fever, rhinorrhea and mild cough followed by certain onset of inspiratory stridor. On examination, her vital signs are stable, oxygen saturation level is 99% on room air and chest X-ray shows steeple signs in anteroposterior view of chest (Fig. 3).

CLINICAL MANAGEMENT

This includes the following:

- Avoid excessive agitation and crying—this will increase respiratory distress causing hypoxia and oxygen demand.
- Humidified Oxygen or cool mist can be provided by mask
- Nebulizer treatment with Racepinephrine for patients with moderate to severe respiratory distress.
- Corticosteroids such as Dexamethasone.

DIFFERENTIAL DIAGNOSIS

- Epiglottitis
- Laryngomalacia
- Vocal cord dysfunction
- *Spasmodic croup*: Much less common than infectious croup and is thought to be allergic in origin. There are no preceding or associated symptoms such as fever or rhinorrhea. Spasmodic croup resolves within a few hours without specific treatment.

DISCUSSION

Acute laryngotracheobronchitis (infectious croup) is the most common cause of upper airway obstruction in young children beyond the neonatal period. The majority of cases result from infection with parainfluenza virus, although influenza, adenovirus and respiratory syncytial virus can also cause croup. Typically, a 1-day to 3-day period of low-grade fever, rhinorrhea, and mild cough is followed by the sudden onset of inspiratory stridor and the characteristic barking cough, often during the night. Hypoxia is unusual, even among patients brought to the emergency department in respiratory distress. Infectious croup is generally a clinical

diagnosis. Subglottic narrowing (the "steeple sign") may or may not be appreciated on an anteroposterior chest radiograph (Figs 2 to 4). Administration of systemic steroids such as Dexamethasone results in significant improvement within hours. For moderate to severe cases, suggested dose is 0.6 mg/kg IV/IM/PO, max dose is upto 10 mg/daily. Nebulized epinephrine is reserved for the patient with moderate-to-severe respiratory distress.

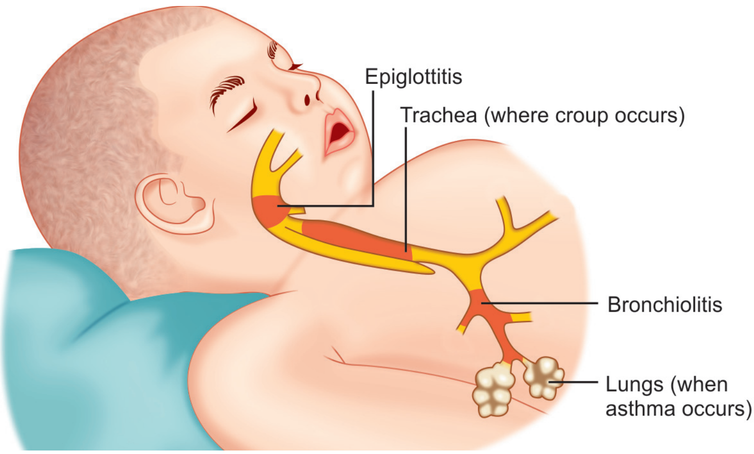


Fig. 1: Location of airway obstruction

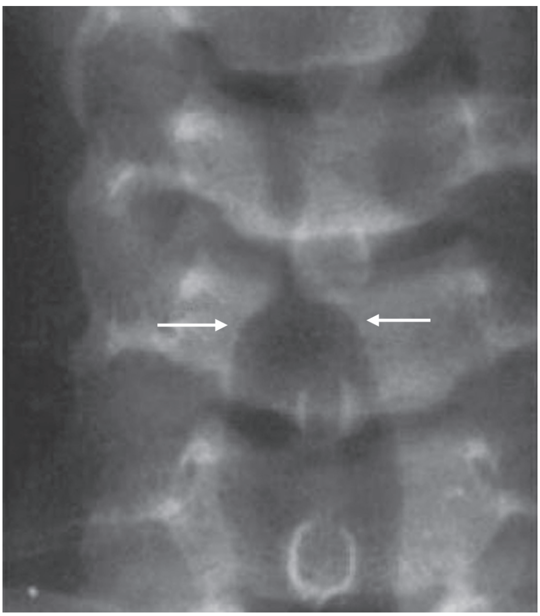


Fig. 2: Normal anteroposterior radiograph of upper airway, with the normal appearance of the subglottic region (arrows)

Fig. 3: Anteroposterior radiograph of the upper airway of a patient with croup. The subglottic tracheal narrowing produces an inverted V appearance known as "steeple sign" (arrows)

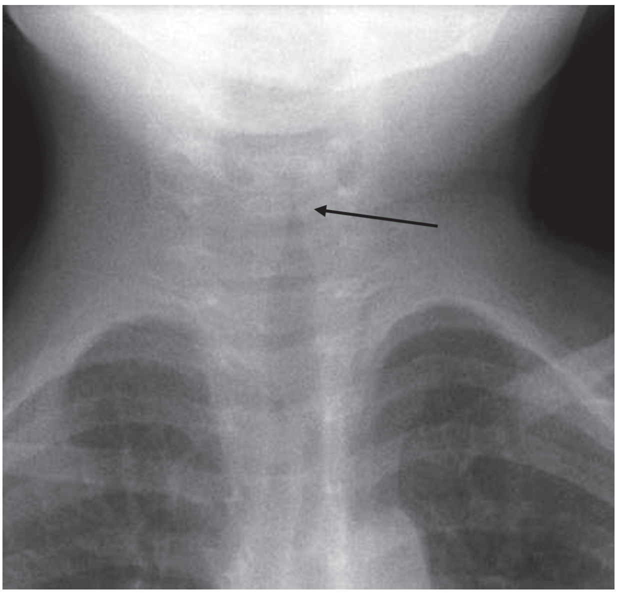


Fig. 4: The steeple sign as seen on an AP neck X-ray of a child with croup
Source: From Wikipedia

Case Study 74: Febrile Seizures

CASE HISTORY

A 2-year-old boy is brought to the emergency department by his mother after she witnessed him having an episode of "full body shaking". The child has had symptoms of an upper respiratory viral infection for the last 2–3 days. She reports that the shaking lasted approximately 5 minutes and the child was very sleepy afterward. On physical examination, the patient has vital signs of temperature 102.4°F, pulse rate 79 beats/minute, respiratory rate 18 breaths/minute and blood pressure 110/65 mm Hg. He is sleepy but arousable, and neurological examination reveals no focal deficits and no meningeal signs. Laboratory studies show a normal complete blood count and electrolytes. Blood glucose level is 100 mg/dL. He is diagnosed with a febrile seizure.

FEBRILE SEIZURES

Febrile seizures are common in children between the ages of 6 months and 5 years. They often occur with temperatures over 39°C but are not related to the actual temperature, rather to the rate of rise of the temperature. There are two types of febrile seizures: simple and complex (Table 1). A simple febrile seizure is one which lasts less than 15 minutes, does not recur within 24 hours, is generalized and occurs in children less than 6 years of age with no underlying neurological disorders. If any of these conditions are not met, then it is categorized as a complex febrile seizure. The children should have additional workup including computed tomography or lumbar puncture and possible hospital admission for observation. Any child with a toxic appearance, meningeal signs, an abnormality on neurologic examination or underlying brain abnormality should not be assumed to have a febrile seizure. Any child who is found to

Table 1: Comparison between simple febrile seizure and complex febrile seizure.[1]

Simple febrile seizure	*Complex febrile seizure*
Lasts less than 15 minutes	Lasts 15 minutes or longer
Occurs once in a 24-hour period	Occurs more than once in a 24-hour period
Generalized	Focal
No previous neurologic problems	Patient has known neurologic problems, such as cerebral palsy

have meningitis during workup should have the seizure attributed to the infection, not to the fever.

Differential Diagnosis of Seizures in Children

- Fever
- Epidural and subdural infections
- Meningitis or encephalitis
- Epidural hematoma
- Sepsis or bacteremia
- Epilepsy

Management

The basis of management of a child who has had a febrile seizure is antipyretic therapy, both during the current illness, as well as during future febrile illnesses. Anticonvulsant therapy is not recommended for children who have experienced febrile seizures, even if they have occurred more than once. Anticonvulsants have serious risks and there is no evidence that children who are given anticonvulsants after the first febrile seizure are at any decreased risk from having another one. It is important to educate parents and caregivers on fever prevention and seizure safety.

Prognosis

One-third of children with a febrile seizure will have another seizure during a subsequent febrile event. Risk factors for recurrent episodes include younger age, family history of febrile seizures, short duration of the fever and a relatively low fever at the initiation of the seizure. There is a small increase in the risk of epilepsy in later life in children who have had a febrile seizure. This risk is greatest in those with a family history of epilepsy, complex febrile seizures and any developmental abnormality.

REFERENCE

1. Millar JS. Evaluation and treatment of the child with febrile seizure. Am Fam Physician. 2006;73(10):1761-4. [online] Available from http://www.aafp.org/afp/2006/0515/p1761 html [Accessed 2012].

Case Study 75: Near Drowning

"Look before you leap"
"I was much further out than you thought, and not waving but drowning"
"Drown not thyself to save a drowning man"
Coast guards are not in place in many developing countries, which leads to the deaths of many children and young adult. These deaths are preventable by the government implementing laws and its citizens respecting the laws. If there are no guards present, then swimming should not be permissible.

—Badar M Zaheer

CASE HISTORY

A 4-year-old boy is brought to the emergency room by ambulance after being found in a backyard pool. The boy had been playing in the backyard unsupervised and fell into the pool. On arrival he is intubated and vital signs are temperature of 95°F, pulse rate 35 beats/minute, respiratory rate 16 breaths/minute (mechanically) and blood pressure 70/40 mm Hg. His Glasgow Coma Score (GCS) is 9.

DISCUSSION

Clinical Presentation

There are two types of drowning: Dry drowning and Wet drowning (Figs 1 to 4). Dry drowning is caused by laryngospasm, which then causes hypoxemia and neurological insult. This accounts for upto 20% of submersion injuries. Wet drowning is due to the aspiration of water into the lungs causing a washout of surfactant, diminished alveolar gas exchange, atelectasis and a ventilation-perfusion mismatch. This leads to noncardiac pulmonary edema with moderate to severe aspiration. Physical examination may reveal clear lungs, wheezes, rhonchi or rales, and mental status may range from normal to comatose. Hypothermia can be seen even in warm water submersions.

Diagnosis

The diagnosis of drowning is usually obvious but it is important to look for other injuries. Spinal cord injuries can occur with diving or surfing injuries or boating accidents. Other conditions including syncope, hypoglycemia, underlying heart disease such as myocardial infarction or dysrhythmias, have been linked to drowning. Laboratory testing may reveal metabolic acidosis and electrolyte abnormalities. There is associated renal injury from the hypoxemia. You may also see hemoglobinuria or myoglobinuria.

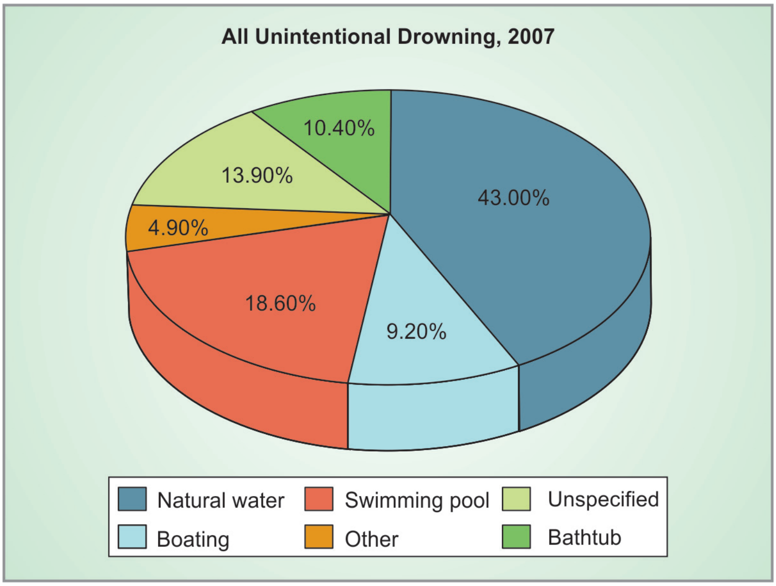


Fig. 1: Drowning Happens Quickly—Learn How to Reduce Your Risk. About 10 people die every day from unintentional drowning. Of these, two are children 14 or younger. Learning the risks and taking safety precautions are proven ways to prevent drowning injuries and deaths

Massive hemolysis can occur with large volumes of aspiration of fresh water, but disseminated intravascular coagulation (DIC) is rare. Necessary tests include a chest X-ray and arterial blood gas analysis. The chest X-ray often shows generalized pulmonary edema or perihilar infiltrates or it may be normal. The arterial blood gas is necessary because chest radiograph findings often do not correlate with arterial pO_2 and oxygen saturation, and metabolic acidosis must be assessed.

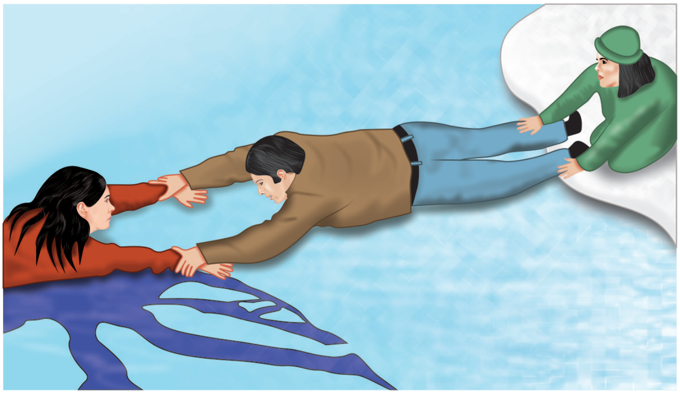


Fig. 2: If a person falls through ice, and there is more than one person on solid ground, form a chain of bodies from a secure location out to the fallen person

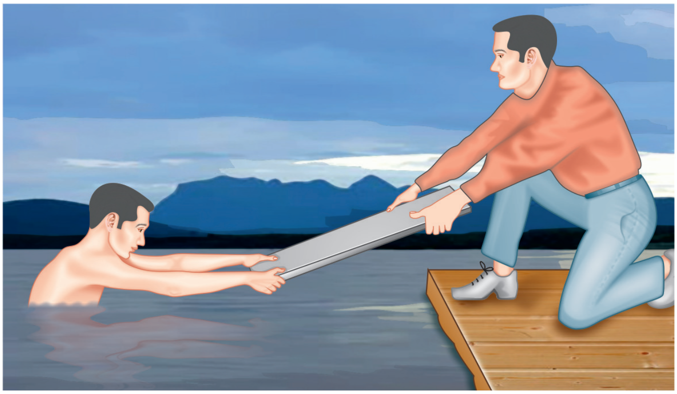


Fig. 3: If the victim is in deep or dangerous water but there is a dock to stand on, try a reaching assist with a long, sturdy object

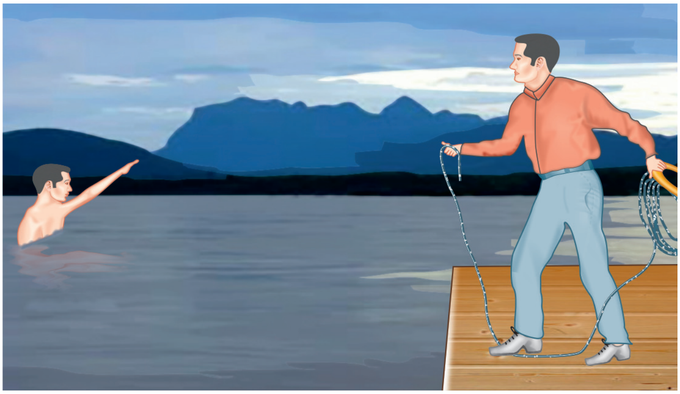


Fig. 4: If the water is too deep or dangerous to enter or if the victim is too far out to reach with a long object, a throwing assist may be wisest

Pulmonary Treatment

The first step is assessing airway, ventilation and oxygenation status along with cervical spine stabilization in the scenario of diving accidents, multiple trauma or unknown mechanisms. If hypothermia is present, the patient should be given warmed intravenous normal saline and warming adjuncts such as overhead warmers or heating blankets. Core temperature must be monitored closely. Victims can then be divided into two categories based on their GCS. Patients with a GCS of at least 14 should be given supplemental O_2 to maintain saturation level above 95%. They may be discharged home after being observed for 4–6 hours as long as pulmonary and neurological examinations and O_2 saturation levels return to normal. If the patient still requires oxygen or has an abnormal examination after 4–6 hours, they should be admitted. Patients with a GCS less than 14 should also be given supplemental O_2, and intubation with mechanical ventilation should be considered if PaO_2 cannot be maintained above 60 mm Hg in adults or 80 mm Hg in children despite high flow oxygen. Pediatric victims of fresh water drowning can sometimes develop dilutional hyponatremia and seizures. These can be controlled by correcting the electrolyte abnormality. The development of pneumonia is rare so prophylactic antibiotics are not indicated as part of treatment.

Neurologic Treatment

"Brain resuscitation" using mannitol, loop diuretics, hypertonic saline, fluid resuscitation, mechanical hyperventilation, controlled hypothermia, barbiturate coma and intracranial pressure monitoring have been attempted but have shown no benefit. Asystole outside of the hospital is a poor prognostic sign in pediatric warm water submersion injuries but there have been reports of neurologic recovery in these situations. If submersion and transfer time were both short, the patient should undergo vigorous attempts at resuscitation. Continuous vasopressor infusion may be necessary in the postresuscitation phase and consideration should be given for withholding resuscitation efforts in patients with prolonged submersion and transport time. There have been reports of full neurologic recovery in near drowning cases in adults and children even after asystole in icy water submersions. Hypothermic victims of cold water drowning in cardiac arrest should undergo prolonged and aggressive resuscitation until they are normothermic and not viable.

Article

Injury Death in Children Ages One to Four

Time (5/18, Rochman) reports, "Drowning is the leading cause of injury death in children ages 1–4 years, according to a Centers for Disease Control and Prevention (CDC) report released Thursday". While, "drowning rates in the US have declined children between the ages of 1 and 4 years have the highest rate of both fatal and nonfatal drowning with 50% of fatal incidents occurring in swimming pools". The report is based on "death certificate data from the National Vital Statistics System and injury data from the National Electronic Injury Surveillance System—All Injury Program (NEISS-AIP) for the years 2005–2009".

HealthDay (5/18, Reinberg) reports, "Males are victims four times as often as females". In the years covered, "more than 3,800 people of all ages drowned annually nationwide".

WebMD (5/18, Nierenberg) reports, "Children under 4 years accounted for nearly 53% of emergency visits for drowning-related injuries, while children aged 5–14 years were responsible for almost 18% of them".

Take Action to Reduce Risks

Learn to swim. Formal swimming lessons can reduce the risk of drowning by as much as 88% among young children aged 1–4 years, who are at greatest risk of drowning.[2] However, even when children have had formal swimming lessons, constant, careful supervision when in the water, and barriers to prevent unsupervised access are necessary to prevent drowning.

Closely watch swimmers in or around the water. Designate a responsible adult who can swim and knows CPR to watch swimmers in or around water—even when lifeguards are present. That adult should not be involved in any other distracting activity (such as reading, or talking on the phone) while watching children.

Learn cardiopulmonary resuscitation (CPR). In the time it might take for lifeguards or paramedics to arrive, your CPR skills could save someone's life.

Fence it off. Barriers to pool access should be used to help prevent young children from gaining access to the pool area without caregivers' awareness when they aren't supposed to be swimming. Pool fences should completely separate the house and play area from the pool, be at least 4-feet high, and have self-closing and self-latching gates that open outward, with latches that are out of the reach of children.

Use the Buddy System. Regardless of your age, always swim with a buddy.

Look for lifeguards. Select swimming sites that have lifeguards whenever possible.

Heed warning flags. Know the meaning of and obey warnings represented by colored beach flags which may vary from one beach to another.

Know the terrain. Be aware of and avoid drop-offs and hidden obstacles in natural water sites. Always enter water feet first.

Avoid rip currents. Watch for dangerous waves and signs of rip currents, like water that is discolored and choppy, foamy, or filled with debris and moving in a channel away from shore. If you are caught in a rip current, swim parallel to shore; once free of the current, swim diagonally toward shore. More information about rip currents.

Use approved life jackets. Do not use air-filled or foam toys, such as "water wings", "noodles", or inner-tubes, in place of life jackets. These toys are not designed to keep swimmers safe.

Avoid alcohol. Avoid drinking alcohol before or during swimming, boating, or water skiing. Don't drink alcohol while supervising children.

Don't hyperventilate. Swimmers should never hyperventilate before swimming underwater or try to hold their breath for long periods of time.

This can cause them to pass out (sometimes called "shallow water blackout") and drown.

CHAPTER

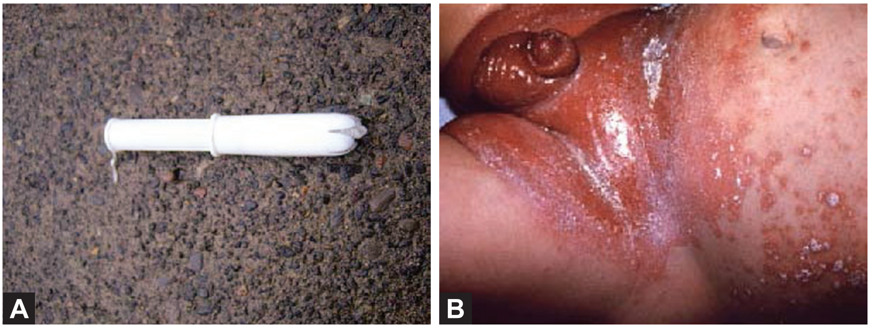
12

Obstetrics and Gynecology

Case Study 76: Toxic Shock Syndrome

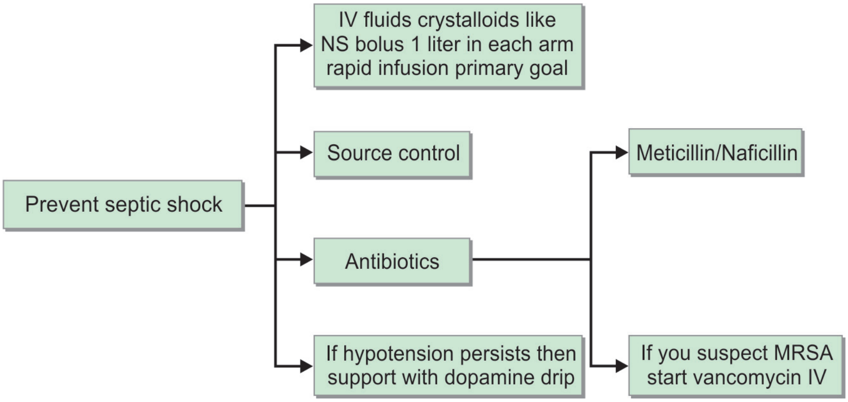
CASE HISTORY

A 30-year-old female is brought to emergency room (ER) for feeling exhausted, tired, weak, nauseous and vomiting for 3 days and has a fever of 103°F with body aches. On physical examination, patient has a blood pressure (BP) of 60/40 mm Hg. She is toxic looking, lethargic and confused. Diffuse rash noticed (Figs 1A and B). On heart examination, she is tachycardic. Lungs have crackles on the bilateral bases. Rest of the examination is normal. Resuscitative measures are taken immediately. Intravenous (IV) fluids started with 0.9% normal saline (NS) bolus through 18G cannulae in each arm in the antecubital vein. After fluid resuscitation, blood cultures are drawn and IV methicillin and nafcillin are started (Flow chart 1 and Table 1). After the bolus, blood pressure improved and the patient is now more alert, coherent and widely awake.



Figs 1A and B: (A) Forgotten tampon; (B) Typical rash of toxic shock syndrome (TSS)
Courtesy: Stephanie Williford Chief Executive Officer, EB Medicine

Flow chart 1: Goals of treatment



(IV: Intravenous; NS: Normal saline; MRSA: Methicillin-resistant Staphylococcus aureus)

Table 1: Toxic shock syndrome: case definition
1. Fever greater than 38.9°C
2. Diffuse macular erythroderma
3. Desquamation 1–2 weeks after onset of illness, especially on palms and soles
4. Hypotension (systolic blood pressure less than fifth percentile)
5. Involvement of three or more organ systems:
• Gastrointestinal (vomiting or diarrhea)
• Muscular (severe myalgia or CPK greater than two times normal)
• Mucous membranes (vaginal, oropharyngeal, or conjunctival hyperemia)
• Renal (BUN or creatinine greater than two times normal)
• Hepatic (total bilirubin, SGOT or SGPT greater than two times normal)
• Hematologic (platelets less than 100,000)
• Central nervous system (altered mental status without focal neurologic signs)
6. Negative results on the following tests:
• Throat, CSF cultures
• Serologic tests for rocky mountain spotted fever or measles.

(CPK: Creatine phosphokinase; BUN: Blood urea nitrogen; SGOT: Serum glutamic-oxaloacetic transaminase; SGPT: Serum glutamic-pyruvic transaminase; CSF: Cerebrospinal fluid)

DISCUSSION

Treatment plan: Treatment plan is discussed in Figure 2. The main goal for treating toxic shock syndrome is to prevent septic shock. Early introduction of IV fluids and antibiotics are necessary.

ADDITIONAL READING

1. www.cdc.gov/ncidod/dbmd/diseaseinfo/toxicshock_t.htm
CDC's National Notifiable Diseases Surveillance System (NNDSS)

Case Study 77: Placenta Previa

CASE HISTORY

A 32-year-old gravida 6, parity 5 (G6P5) female is presented to the emergency room (ER) with painless vaginal bleeding. This started early in the morning of presentation. It is profuse and very bright red in color (Fig. 1). Patient had some spotting a month ago after intercourse. On examination in the ER, vitals included temperature 99°F, pulse rate 80 beats/minute, blood pressure 110/60 mm Hg. Fetal heart rate monitor shows 140–150 beats/minute.

DISCUSSION

Placenta previa is defined as the implantation of the placenta over the cervical os. There are three types: (1) total, (2) partial and (3) marginal.

DIAGNOSTIC WORKUP

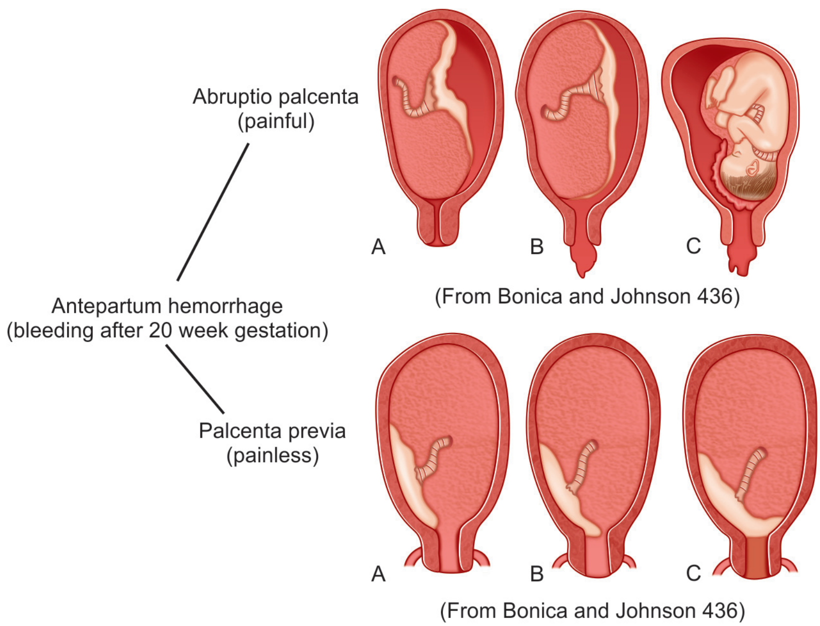


Fig. 1: Types of placenta

MANAGEMENT

- Please do not do any digital or speculum examination when you have a patient who presents with antepartum painless vaginal bleeding.
- Always suspect placenta preevia and rule it out with a transabdominal ultrasound. A transabdominal ultrasound will not always be able to detect the location of the placenta. In such cases, a transvaginal ultrasound is necessary by an experienced physician.
- The first episode of vaginal bleeding does not cause sufficient concern to necessitate emergent delivery. In such cases, with a preterm gestation in placenta preevia, patients are usually observed on bed rest. This is done with the expectation that time may be gained for fetal maturity. Amniocentesis is done to establish fetal lung maturity.
- If the fetal lungs appear to be mature, then delivery by C-section will be scheduled.

CAUTION

Digital examination should not be performed prior to ultrasound.

ADDITIONAL READING

1. Eugene Toy 2009. Case Files Obstetrics & Gynecology, 3rd Edition.

Case Study 78: Ectopic Pregnancy

CASE HISTORY

The patient is a 26-year-old gravida 3, parity 4 (G3P4) female with history of past ectopic pregnancy (EP) who presents to the emergency room with severe right-sided abdominal pain for 3 days. The pain is 10/10, sharp, and localized to the right lower abdomen without radiation. She reports associated nausea, but no vomiting. Tylenol did not relieve the pain. Patient denies any light headedness or loss of consciousness. She states that this pain feels exactly as the pain she had with her previous EP which she believes was located on the left side. Patient denies vaginal bleeding or discharge. She did not take a pregnancy test at home, but she is sexually active with her husband and is not currently using birth control because she wishes to become pregnant. No history of sexually transmitted diseases (STDs) in the past is noted. Last menstrual period (LMP) was approximately 6 weeks ago. No significant past medical history is noted. Surgical history is significant for two cesarean (C)-sections and laparoscopic removal of the EP with preservation of the uterine tubes. Patient does not drink, smoke, or use any recreational drugs. On physical examination, patient is afebrile with blood pressure of 100/70 mm Hg, pulse rate 120 beats/minute, respiratory rate 20 breaths/minute and saturation is 99% on room air. On abdominal examination, patient is severely tender to light palpation in the right lower quadrant. No rebound tenderness, or percussion tenderness, but positive Rovsing's sign noticed. Urine pregnancy test is positive. IV Fluids, Zofran® and pain medications are started.[1] Two large bore intravenous lines (IV) are placed. Pelvic examination was positive for cervical motion tenderness (CMT), but no evidence of vaginal bleeding or discharge was found. Transvaginal ultrasound revealed a large sac-like structure on the right adnexa. Ultrasound revealed only gestational sac. Beta-human chorionic gonadotropin (β-hCG) was 3,000 mIU/mL. Obstetrician/gynecologist (ob/gyn) was consulted for further management of likely ectopic pregnancy. Further ob/gyn transvaginal ultrasound confirmed EP on the right side measuring 4 × 5 cm (Fig. 1). Patient was taken to the operating room (OR) for laparoscopic removal.

DISCUSSION

In 20% of cases, ectopic pregnancies are ruptured at the time of presentation (Fig. 2). Risk factors for EP include history of pelvic inflammatory disease (PID), surgical procedure of the Fallopian tubes including tubal

ligations, previous EP, intrauterine device (IUD) use, assisted reproductive technologies, abdominal pain, vaginal bleeding and amenorrhea. Only 90% of women with EP complain of abdominal pain, 80% have vaginal bleeding, 70% have a history of amenorrhea. Pain is described as sudden, lateral, extreme and diffuse. Vaginal bleeding is usually light. Heavy bleeding is common with abortion or other complications of pregnancy.

Abdominal examination shows signs of localized or diffuse tenderness with or without peritoneal signs. Pelvic examination findings may be normal, but most often shows CMT and adnexal tenderness, with or without a mass. The patient may possibly have an enlarged uterus. Fetal heart tones are rarely audible.

In order to make a diagnosis, transvaginal ultrasound is the test of choice. A progesterone level of 5 ng/mL or lower with an empty uterus is highly suggestive of EP, but it cannot be used to exclude EP. A high β-hCG level greater than 6,000 mIU/mL with empty uterus is suggestive of EP. If hCG is low and less than 1,000 mIU/mL, then the pregnancy may be intrauterine or ectopic, but not able to be visualized on ultrasound. In 2 days, a repeat β-hCG must be performed in these situations.

The emergency department management depends on vital signs, physical examination findings and symptoms. Two large bore IV lines are started in case rapid infusion of crystalloids and/or packed red blood cells (pRBCs) are needed. Urine pregnancy test should always be obtained first. If identified, ob/gyn consultation is necessary for further workup. Blood is typically drawn for complete blood count (CBC), blood type, Rhesus (Rh) factor, β-hCG and serum electrolytes. For unstable patients, the diagnostic workup includes transvaginal ultrasound. Patients with indeterminate ultrasound and hCG less than 1,000 mIU/mL can be discharged with ectopic precautions and follow up in 2 days.

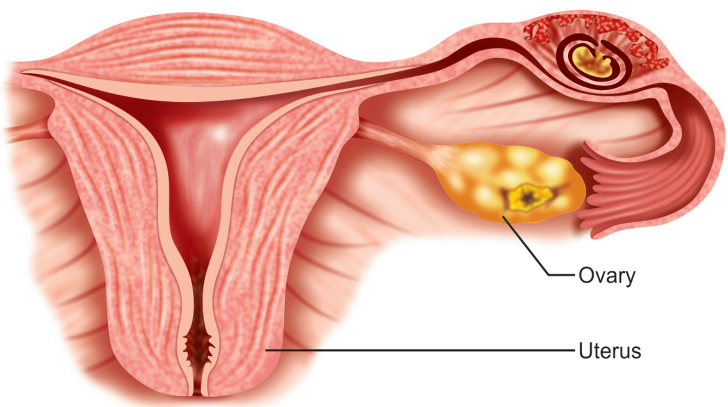


Fig. 1: Ectopic pregnancy

Fig. 2: Cullen's sign—sign of ruptured ectopic
Photograph by: Herbert L Fred, MD and Hendrik A van Dijk

Unless the medical team is very vigilant and maintain a high index of suspicion, it is easy to miss ectopic pregnancies.

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1. Dunn RJ, Dilley S, Brookes JG, et al. (2013). The Emergency Medicine Manual [Online] Available from www.emergencymedicinemanual.com/ [Accessed September, 2013].

CHAPTER

13

Ear, Nose and Throat Emergencies

Case Study 79: Epistaxis

"Not every epistaxis is just a bloody nose."

—Badar M Zaheer

CASE HISTORY

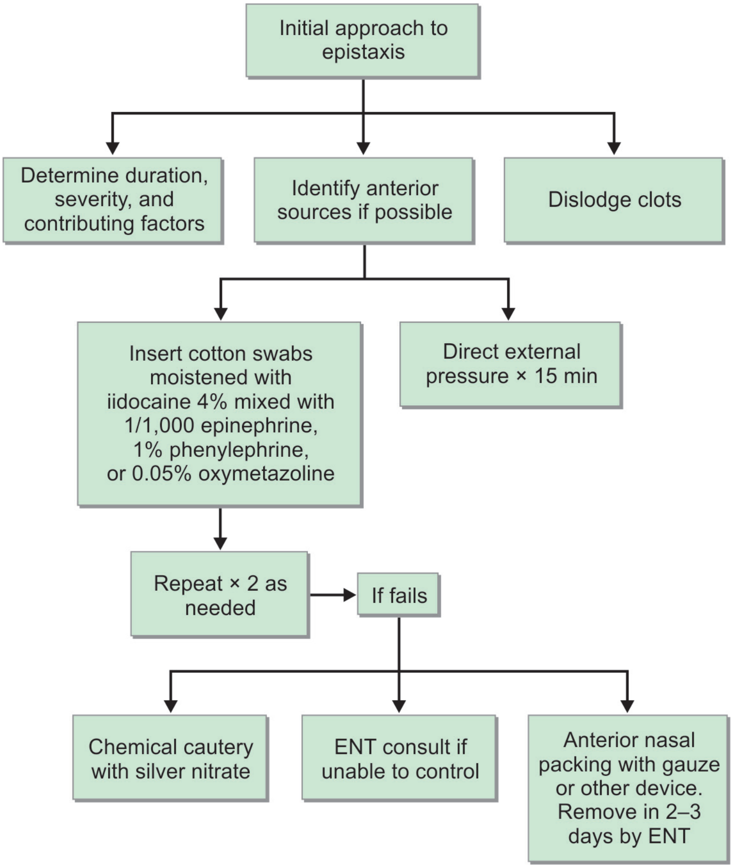
A 10-year-old boy comes in emergency department (ED) for recurrent nosebleeds. He has a history of allergies and has been taking Flonase regularly to control his sinusitis. He presents to the ED overnight when the father can no longer control the bleeding on his own. The physician locates a source near the anterior nare, but the bleeding does not stop after administering cotton swabs moistened with lidocaine several times. Finally, the bleeding is stopped after administering silver nitrate. Patient is instructed to follow-up with an ENT physician in 3 days.

DISCUSSION

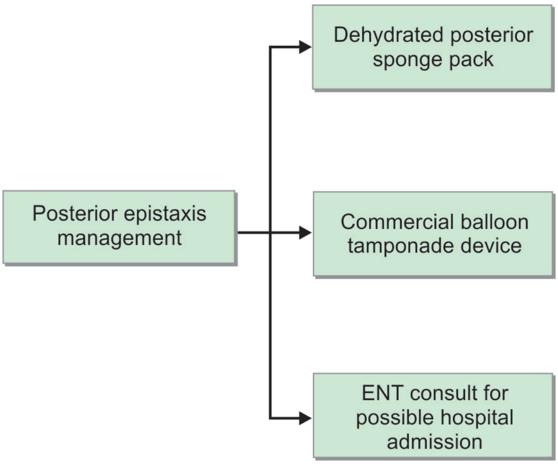
The key of managing epistaxis is to determine whether the bleeding is anterior or posterior. Usually posterior bleeding is identified by lack of visualization of anterior source, bleeding from both nares or drainage into posterior pharynx after controlling anterior source (Flow chart 1 and 2). The majority are anterior bleeds which come from Kiesselbach's plexus (Fig. 1). These patients are usually children or young adults. Risk factors include trauma, epistaxis digitorum, winter syndrome, allergies, irritants such as cocaine or nasal sprays, and pregnancy. Posterior bleeding accounts for only 10% and is usually present in the elderly. Anatomically, these bleeds are arterial in nature. Etiologies include coagulopathy, atherosclerosis, neoplasm and questionable hypertension.

Anterior epistaxis can be treated with anterior packing balloons, but some patients are uncomfortable with this option. Alternative treatment can be fashioned from available materials in the ER. An absorbable hemostatic sponge can be coated with bacitracin, inserted into the nare,

Flow chart 1: Management of epistaxis



Flow chart 2: Management of posterior epistaxis



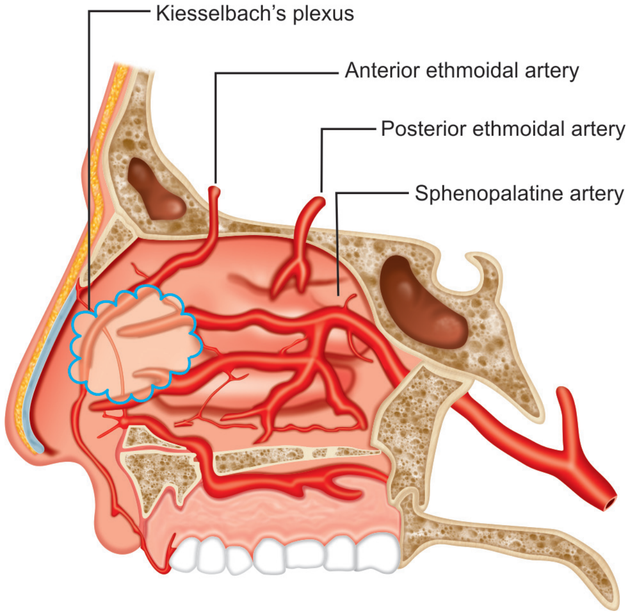


Fig. 1: Nasal vascular anatomy
Courtesy: Goralnick E. (2012). Posterior epistaxis nasal pack. [online] Available from emedicine. medscape.com/article/80545-overview. [Accessed September, 2013].

and affixed to the septum using tissue adhesive. Tongue depressors can be taped together two-third from the distal end and used to provide compression.

Complications of nasal packing include dislodgement of the pack, recurrent bleeding including septal hematomas, abscesses, neurogenic syncope, pressure necrosis, sinusitis and toxic shock syndrome. If pack is placed, use antibiotic prophylaxis such as cephalexin 250–500 mg PO q 6 h or amoxicillin/clavulanate 250/150 mg PO q 8 h. However if the patient is allergic to penicillin, you may use clindamycin or trimethoprim or sulfamethoxazole. It is also important to consider comorbid CSF rhinorrhea (Fig. 2). Clues which should raise a clinician's level of suspicion for this diagnosis include unilateral nasal discharge and loss of the sense of smell, however the absence of these signs and symptoms does not rule out CSF rhinorrhea. A positive "halo sign" can be found if the blood has mixed with another fluid such as CSF, tears, or saliva. If a CSF leak is suspected, high-resolution CT and CT cisternography should be obtained.

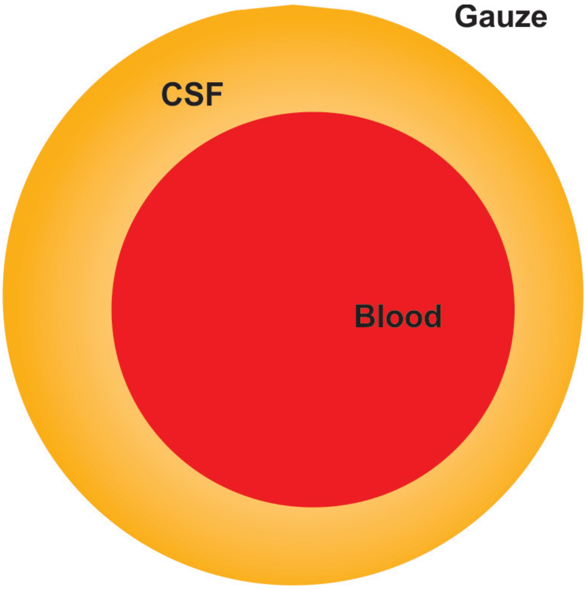


Fig. 2: A positive halo sign on the background of tissue paper
Courtesy: Dr Badar M Zaheer

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2. Kucik CJ, Clenney T. Management of epistaxis. Am Fam Physician. 2005;71(2):305-11. Available from www.aafp.org/afp/2005/0115/p305.pdf.
3. Viduchich R, Blanda M, Gerson L. Posterior epistaxis: clinical features and acute complications. Ann Emerg Med. 1995;25(5):592-6. http://download.journals.elsevierhealth.com/pdfs/journals/0196-0644/ PIIS0196064495701699.pdf.
4. Badar Zaheer MD. Halo sign demonstrated on a piece of gauze.

Case Study 80: Nasal Fractures

CASE HISTORY

A 13-year-old girl is presented to the emergency department (ED) with swelling, tenderness, crepitus and nose deformity. On examination, vital signs are stable. X-ray of the nasal bone shows fracture of the nasal bones. ENT examination shows collection of bluish-filled sacs and grape-like cluster on the left nasal side. Patient is initially treated with ice pack and ibuprofen, and then referred to ENT department. She returns with worsening pain, difficulty in breathing and drainage from the site of the bluish-filled sac. Patient has an abscess at the site of the septal hematoma, which is drained with incision and drainage. Pain significantly decreases after the procedure and patient is discharged with a 7-day course of antibiotics.

DISCUSSION

This patient has a fracture of the nasal bones with the complication of a septal hematoma as indicated by the bluish-filled sac in the left nasal side (Fig. 1). Treatment is generally conservative with ice packs, ibuprofen, nasal packing if necessary and referral to ENT.[1] Despite conservative treatment the patient presented with difficulty in breathing and swelling of the left nasal septum suggestive of a superimposed infection on the left nasal septal hematoma. Incision and drainage is performed with lidocaine nasal spray, which is used to anesthetize the wound area. Prophylactic antibiotics are prescribed as indicated.

Nasal fracture is a clinical diagnosis if the injury mechanism, swelling, tenderness, crepitus, gross deformity and periorbital ecchymosis are seen.[2] Other symptoms include epistaxis, rhinitis, nasal vestibular stenosis and airway obstruction. Radiological study is usually not required in the ED.

Follow-up should be done in 2–5 days for reassessment. The nose should be evaluated for a septal hematoma because if left untreated, it may result in abscess formation or necrosis of the nasal septum. A septal hematoma is a collection of blood beneath the perichondrium. It may appear as a bluish, fluid-filled sac on nasal septum. Local incision and drainage followed by anterior nasal packing is the form of treatment. If a cribriform plate injury is suspected, computed tomography scans should be performed followed by immediate neurological consultation.

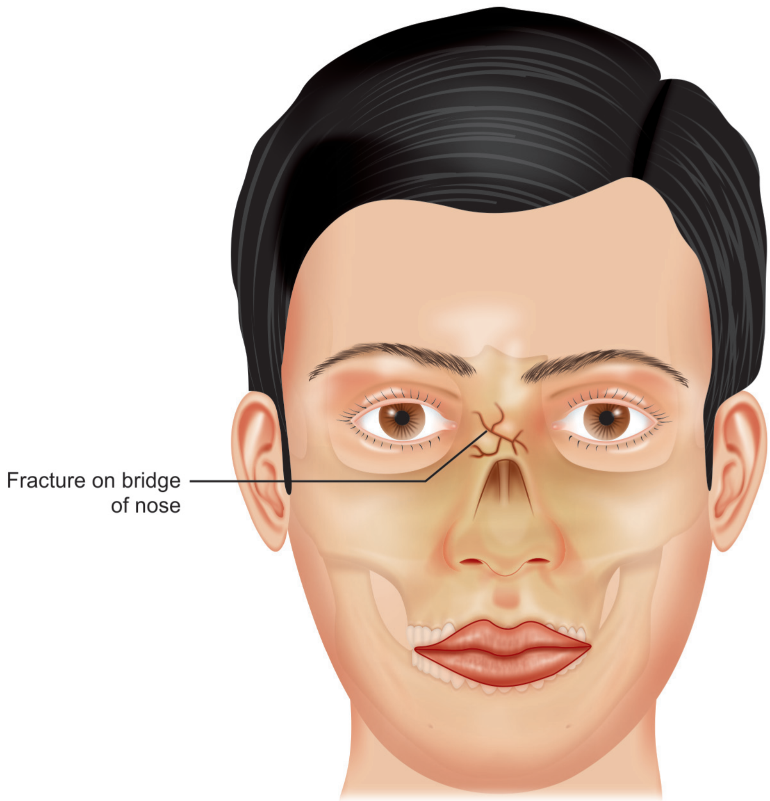

Fig. 1: Nasal fracture
Courtesy: Vorvick LJ. (2011). Nasal fracture. [online] Available from www.nlm.nih.gov/medlineplus/ency/imagepages/8873.htm. [Accessed September, 2012].

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2. Glaspy JN. Nasal fractures. Emergency Medicine Manual, 6th edition. New York: McGraw Hill; 2004. p. 732.

Case Study 81: Nasal Foreign Bodies

CASE HISTORY

A 6-year-old girl is brought to the clinic by her parents. The parents found unilateral, foul smelling purulent discharge from her nose. Her younger sister said she was playing with beads and inserted one in her nose. Child complains of difficulty breathing from one nostril and feels something in the nose. Child complains of irritation and pain upon assessment.

DIAGNOSIS

Nasal foreign body

MANAGEMENT

Topical anesthesia is given, which is followed by removal of the foreign body with a probe.

Do not attempt to search the nose with cotton swab or other tools for risk of pushing object further in.

DISCUSSION

Patients with nasal foreign bodies usually present with unilateral nasal obstruction, foul rhinorrhea, or persistent unilateral epistaxis. Topical vasoconstrictors and anesthesia should be used, and the foreign body should then be removed. Hooked probes, balloon-tipped or suction catheters and/or forceps are the standard tools used to remove the foreign body (Fig. 1). For pediatric patients, the nonaffected nostril should be occluded with a finger and positive air pressure should be applied to the patient's mouth. For any unsuccessful removal, an ENT consultation should be sought.

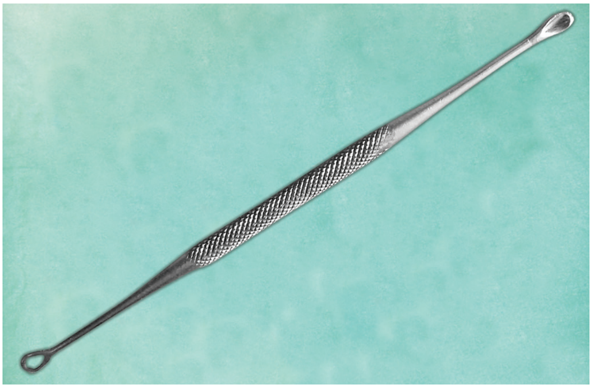

Fig. 1: Nasal foreign body removal hook used in ENT practice
Source: Reprinted with permission from entinstruments.blogspot.com

Case Study 82: Retropharyngeal Abscess

CASE HISTORY

A 40-year-old male is presented to the emergency department (ED) at 2 AM with symptoms of sore throat, difficulty in swallowing and trismus. Patient initially had a sore throat and difficulty in swallowing over the past 2 weeks, but is now getting worse. He can no longer open his jaw and he continues to have high fevers at home up to 104°F. On presentation to the ED, the patient is ill-appearing. Vitals are temperature 39.5°C, blood pressure 100/70 mm Hg, pulse rate 120 beats/minute, respiratory rate 18 beats/minute and O_2 saturation level 100%. Computed tomography (CT) scan of neck is done and reveals paravertebral abscess. Physical examination shows tender cervical lymphadenopathy, neck swelling and torticollis. Movement of the trachea or larynx side to side (tracheal "rock") is painful.

DISCUSSION

Retropharyngeal abscesses occur anterior to the prevertebral space and posterior to the pharynx (Fig. 1). These generally occur in children less than 4 years of age, but may occur in adults as well.

Symptoms include pain, dysphagia, dyspnea, fever, muffled voice, stridor and neck stiffness. Patients typically present in a supine position with slight neck extension to improve dyspnea. Physical findings include tender cervical lymphadenopathy, neck swelling, torticollis, pharyngeal erythema and edema. Swelling of retropharyngeal space and thickening of the prevertebral space may be evident on X-ray of lateral soft tissue of the neck (Fig. 2). Further evaluation to define the abscess as well differentiation from cellulitis can be done by CT scan. Complications of retropharyngeal abscess include mediastinitis.

Due to the anatomic and pathophysiologic differences, retropharyngeal abscess is more localized in children than adults.

Differential Diagnosis

X-ray of the neck, lateral soft tissue, will show thickening in the prevertebral space. CT scan of the neck, with enhanced IV contrast, will help us to differentiate between cellulitis and abscess. It also helps us to define the extent of the infections. Cultures taken from the abscess are usually polymicrobial which includes aerobic and anaerobic organisms.

Management

Management should initially focus on airway management. ENT consultation should be obtained for drainage. Antibiotics should be started. Regime for adults may include clindamycin 600–900 mg intravenously (IV) q 8 hours or ampicillin-sulbactam 3 g IV q 6 hours.

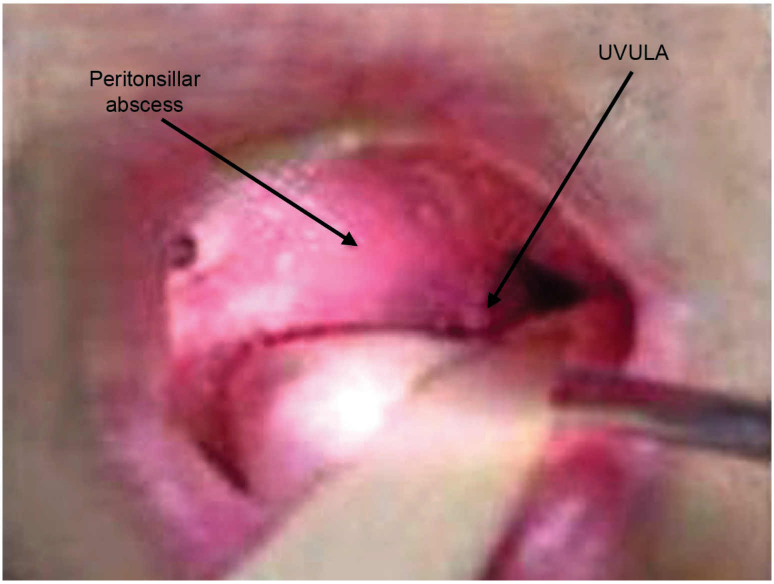

Fig. 1: Peritonsillar abscess
Courtesy: Available from drugster.info/img/ail/1373_1382_2.jpg

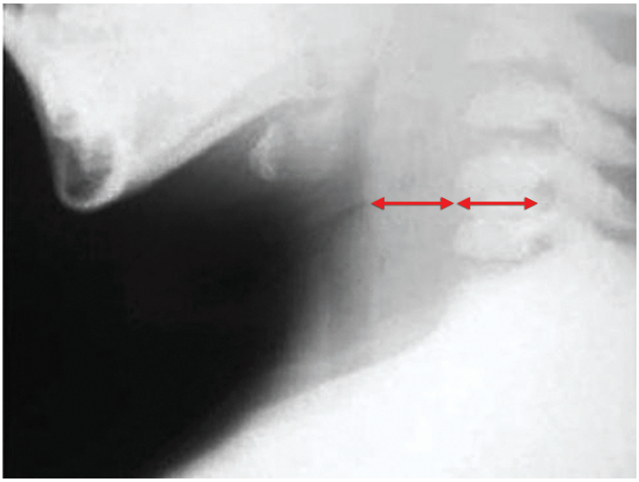

Fig. 2: Retropharyngeal abscess
Courtesy: Acevedo JL. (2011). Pediatric retropharyngeal abscess. [online] Available from reference.medscape.com/features/slideshow/pediatric-respiratory. [Accessed September, 2012].

Case Study 83: Foreign Body/Coin Ingestion

CASE HISTORY

A 3-year-old girl is presented to the emergency department after swallowing a coin. Her mother saw her take the coin off the floor, but was unable to reach her in time before the coin was ingested. She is certain it is a coin and not a battery because she saw the copper color of a penny. The mother does not note any signs of difficulty in breathing or swallowing. The girl appears to be normal, but the mother is concerned that the coin will "get stuck". Vitals are unremarkable including the child being afebrile. On examination, there are no signs of respiratory distress. There is some mild erythema present in the throat. Abdominal examination does not reveal any peritoneal signs. An X-ray is taken and reveals that the coin is now in the child's stomach. Serial abdominal examinations are done, and the child is discharged home with close follow-up by the primary care physician.

DISCUSSION

The majority of foreign body ingestions occur in children, however, they may occur at any age.[1] In adults, foreign body ingestions rate is higher in inmates, toothless adults and psychiatric patients. The foreign body tends to get stuck at sites of narrowing throughout the gastrointestinal (GI) tract. However, once past the pylorus of the stomach, problems do not usually occur.

Symptoms of ingestion may include anxiety, retrosternal discomfort, retching, vomiting, dysphagia, coughing, choking, aspiration and difficulty in swallowing secretions. Physical examination should include the upper airway. Findings in the pediatric population that are supportive of a foreign body ingestion include red throat, palatal abrasion, temperature elevation and peritoneal signs. Either X-ray or laryngoscopy or endoscopy may be used. Be aware that only radiopaque objects will be evident on X-ray. The differentials should include dysphagia, esophageal carcinoma and GI reflux.

Management

Management should include either aspiration of the foreign body or suction of secretions. Abdominal examinations should be monitored closely to watch for signs of perforation. Serial abdominal X-rays may monitor progress of the foreign body through the GI tract. If the type of foreign body is known, treatment can be geared toward the individual

type of foreign body. With food, conservation treatment is safe when the patient is able to manage his or her own secretions. If there is no passage after 12 hours then intervention is deemed necessary. Intervention many include lower esophageal sphincter relaxation with glucagon, nitroglycerin or nifedipine or endoscopy. Avoid proteolytic enzymes as they may cause esophageal perforation. GI consultation will usually be required in the presence of impaction.

For coins, 33% of children will be asymptomatic if the coin is in the esophagus.[2] X-ray is recommended. If in the esophagus, the coin will be in the frontal plane. If in the trachea, the coin will be in the sagittal plane (Fig. 1). If present in the esophagus, the foreign body is generally considered impacted. If lodged in the esophagus, endoscopy is required. If the coin is in the stomach, it will almost always pass on its own.

Battery ingestion is always an emergency situation. Emergent imaging and endoscopy is required except in cases of asymptomatic button batteries that have already passed the esophagus. Button batteries can be conservatively managed unless they are lodged in a fixed location. Burns may occur within 4 hours, and perforation may occur within 6 hours. If a mercury-containing battery has opened, be sure to monitor blood and mercury levels.

Sharp object ingestion management is controversial. An X-ray should be performed to determine location. If longer than 5 cm and wider than 2 cm, it is unlikely that the object will pass the stomach. Pointed objects may cause perforation especially at the ileocecal valve.

Fig. 1: Frontal view of esophageal button (disk) battery; note distinctive double-circle appearance, useful to differentiate a button battery from a coin

Courtesy: Available from emedicine.medscape.com/article/801821-treatment#a1126

Cocaine ingestion is also a consideration. Multiple small bags are often swallowed for drug concealment. Full bowel irrigation has been used successfully, but is not always required. Be careful to monitor vital signs in case the bags burst. Endoscopy should not be performed due to risk of bag rupture. Surgical intervention is safer.

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1. Baines P. Swallowed foreign bodies. Emergency Medicine Manual, 6th edition. New York: McGraw Hill; 2004. pp. 209-11.
2. Conners GP. (2011). Pediatric foreign body ingestion. [online] Available from emedicine.medscape.com/article/801821-treatment#a1126. [Accessed September, 2012].

CHAPTER

14

Hematology/Oncology

Case Study 84: Neutropenic Fever

CASE HISTORY

A 40-year-old woman with history of non-Hodgkin's lymphoma is presented to the emergency department (ED) with fever. Patient has no complaints except occasional weakness and fever this morning while at home. She has not traveled anywhere recently and has been careful to avoid sick contacts. In the ED, she is febrile at temperature 38.4°C, and her other vitals are blood pressure 140/70 mm Hg, pulse rate 95 beats/minute, respiratory rate 18 breaths/minute and O_2 saturation level 96% on room air. Her venous catheter site is without erythema or swelling. Lungs have scattered rhonchi throughout. Remainder of examination is unremarkable. Chest X-ray (CXR) shows no evidence of infiltrate. Absolute neutrophil count (ANC) of 450 cells/mL is revealed. What should be considered in this case?

DISCUSSION

Neutropenic fever is defined as a single fever greater than 38.3°C or a sustained fever greater than 38°C in a person with neutropenia. Neutropenia is defined as having an ANC less than 500 cells/mL. This is a medical emergency with a high-risk of death with ANC less than 100. The potential for death within hours may occur and rapid action is crucial. Because patients without neutrophils are unable to mount a response, there are usually minimal symptoms with a high-risk of rapid deterioration. Patients also on steroids may not mount a febrile response and in fact may become hypothermic and hypotensive. Deterioration may be unexplained in these patients. The fever may be due to viral or bacterial causes.

Differential Diagnosis and Workup

The differential diagnosis for sources of infection includes the central venous catheter site, skin, mouth, sinuses, chest or lung, abdomen, perianal region and central nervous system (brain abscess, encephalitis,

meningitis). Take cultures from all lumens, skin and line sites, sputum, urine and stool for bacterial, fungal and viral cultures. As mentioned previously the patient may have an absence of the usual evidence of infection since neutrophils are required to mount a response. For example, the patient may lack evidence of an infiltrate on CXR with suspected pneumonia.

Workup requires that a complete blood count, chemistry, liver panel, coagulation panel, urine analysis, blood culture and sputum culture should be done.

Emergency Room Care and Disposition

- Antibiotics should be started within 60 minutes empirically.
- Start IV fluids to maintain hemodynamics to prevent the patient from going to septic shock.
- Catheter removal for catheter related infections.

Central venous catheter removal for infections caused by *S aureus*, *P aeruginosa*, Candida spp, or rapidly growing nontuberculous mycobacteria.

Empiric antibiotic therapy should be initiated. In case of persistent or recurrent fever, antifungal agents should be tried.

ADDITIONAL READING

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2. Cleveland Clinic Manual: Infectious Disease Emergencies. p. 710.

Case Study 85: Tumor Lysis Syndrome

CASE HISTORY

A 45-year-old women is presented to the emergency department (ED) after her first round of chemotherapy for her newly diagnosed chronic myelogenous leukemia (CML). Patient reports increasing lethargy, muscle cramps and nausea/vomiting since her chemotherapy. Fluids and Zofran are started. Laboratory tests reveal values of potassium 5.5 mEq/L and serum creatinine 1.8 mg/L. Patient is presumed to be at high risk of tumor lysis syndrome. Aggressive fluid hydration is started, and uric acid, phosphate and calcium levels are ordered.

DISCUSSION

Acute tumor lysis syndrome is a group of metabolic disturbances as a result of the death of rapidly growing tumors. This typically occurs within hours to days after chemotherapy or radiation.[1] Furthermore, this typically occurs after chemotherapy for hematologic malignancies including acute leukemias or high-grade non-Hodgkin's lymphomas. There is increased risk with increased bulk of tumor, and hyperuricemia or renal impairment before chemotherapy. Other risk factors include lactate dehydrogenase levels (> 1,500 U/L), advanced disease with abdominal involvement, pre-existing renal dysfunction, post-treatment renal failure, acidic urine, pre-existing volume depletion and a young age.

Features of acute tumor lysis syndrome include hyperuricemia, high blood lactate level, hyperkalemia, hyperphosphatemia, hypocalcemia secondary to hyperphosphatemia, acute renal failure, cardiac dysrhythmias, neuromuscular symptoms, lactic acidosis and metabolic acidosis. These metabolic abnormalities may result in muscle cramps, tetany, confusion or convulsions and even sudden death.

Management

Management should focus on prevention by identifying high-risk patients. In order to preserve renal function, it is important to recognize renal complications early. Chemotherapy should be delayed until metabolic abnormalities are corrected. Hydration is crucial with rate as high as 4–5 L/day in order to maintain urine volumes of 2–3 L/day unless congestive heart failure is present. Hydration is also important before chemotherapy or radiation therapy. In extreme cases, hemodialysis may be considered. Hemodialysis should be done if serum potassium

is 6 mEq/L, serum uric acid is 10 mg/dL, serum creatinine is 10 mg/dL, serum phosphorus is 10 mg/dL or rapidly rising, with symptomatic hypocalcemia or if there is a need to reduce volume overload. There are multiple ways to reduce uric acid levels. Allopurinol may be used but may not affect levels until 48–72 hours. Rasburicase is rarely used in the ED. Urine may be alkalinized but this is used with caution. Overall, patients have a good prognosis in the absence of renal failure.

REFERENCE

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Case Study 86: Superior Vena Cava Syndrome

CASE HISTORY

A 62-year-old male is presented to the emergency department with left upper extremity and neck swelling, cough, shortness of breath and weight loss for the past several weeks. Cough is productive and sometimes blood streaked. Shortness of breath is exertional and mildly alleviated by his albuterol inhaler. Past history is significant for chronic obstructive pulmonary disease and smoking one pack/day since the age of 17 years. On physical examination, vitals are unremarkable. Remainder of examination is significant for facial fullness, left arm enlargement and distended superficial veins on the chest wall. Chest reveals expiratory wheezing throughout and decreased breath sounds in the left upper lung field.

DISCUSSION

Superior vena cava (SVC) syndrome is commonly caused by malignancy especially lung cancer and lymphoma.[1] Approximately 2–4% of patients with lung cancer develop SVC syndrome, although the incidence is higher in small cell lung cancer. The cause is usually due to masses in the middle or extrinsic mediastinum, right paratracheal or precarinal lymph nodes and tumors extending from the right upper lobe bronchus.

Symptoms

Symptoms typically include facial or neck swelling, arm swelling, dyspnea, cough and dilated chest veins (Fig. 1). Other symptoms may include chest pain, dysphagia, hoarseness, headache, confusion, dizziness or syncope. Red flag signs are stridor suggestive of laryngeal edema and confusion and obtundation suggestive of cerebral edema. Superior vena cava syndrome is only a medical emergency when signs of laryngeal or bronchial or cerebral edema are present, so these are important signs to look for.

Diagnosis

The diagnosis is initially based on clinical signs and symptoms and then confirmed by imaging. Further clarification requires biopsy and imaging. Treatment is based on specific malignancy including staging. Eighty four percent of people will have an abnormal chest X-ray with possible mediastinal widening and pleural effusion. However, computed tomography scans of chest with contrast is the best possible way to

Fig. 1: A person suffering from superior vena cava syndrome having distended veins in the upper chest and arms

look for enlarged paratracheal lymph nodes as well as lung or pleural abnormalities. Venography should be done if there are plans to place a stent.

Management

Management is mostly geared toward symptomatic relief. This includes oxygen support, minimizing hydrostatic pressure in the upper torso by fluid restriction, head elevation and diuretics as well as protecting the airway as necessary. For cerebral edema, consider decreasing intracranial pressure by standard measures. Steroids are controversial, but may be considered as a temporary measure to reduce edema. Definitive therapy includes radiation therapy or chemotherapy to treat malignancy. Endovascular stenting by an interventional radiologist may have immediate relief or within 24–72 hours. This restores venous return in cases with chemotherapy or radiotherapy resistant tumors.[2]

REFERENCES

1. Ugras-Rey S, Watson M. Selected oncologic emergencies, Chapter 121. In: Marx JA, Hockberger RS, Walls RM (Eds). Rosen's Emergency Medicine Concepts and Clinical Practice, 7th edition Philadelphia: Mosby Elsevier; 2009.
2. Wan JF, Bezjak A. Superior vena cava syndrome. Emerg Med Clin North Am. 2009;27(2):243-55.

Case Study 87: Von Willebrand's Disease

CASE HISTORY

A 17-year-old female with known Von Willebrand's disease is presented to the emergency department after a motor vehicle accident. She is currently stable but bleeding is still a major concern.

ETIOLOGY

This disease is a genetic inherited deficiency in Von Willebrand's factor causing defective primary hemostasis between the platelets and the blood vessel walls. There are three major categories of Von Willebrand's deficiency: Type 1 is partial quantitative deficiency, Type 2 is qualitative deficiency and Type 3 is total deficiency.[1]

TREATMENT

Bleeding is a major concern in patients with Von Willebrand's disease as they are unable to form clots to stop major sites of bleeding (Figs 1A and B). The first step in treating a trauma patient with this disease is to administer 10 units of cryoprecipitate as well as fresh frozen plasma as these contain functional Von Willebrand's factor and will thus allow

Figs 1A and B: Girls suffering from Von Willebrand's disease having bleeding from eyes

the patient to stop bleeding. These treatments are usually reserved for trauma scenarios because of the high-risk associated with foreign blood product transfusion.

REFERENCE

1. Pollak ES. (2012). Von Willebrand Disease. [online] Available from emedicine.medscape.com/article/206996-overview. [Accessed September, 2012].

CHAPTER

15

Trauma in Extremes of Age

Case Study 88: Trauma in Elderly

"Respecting your parents and elders is a universal commandment in every religion."

—Badar M Zaheer

CASE HISTORY

A 70-year-old female is presented to the emergency department (ED) after a fall. The patient was walking out to her car when she slipped and fell on the driveway with an outstretched hand. A neighbor stepped out just in time to see the fall and immediately called 911, for emergency medical help. The neighbor reported that she broke most of the fall with her hand, but she did strike her head on the pavement. However, she never lost consciousness. On presentation to the ED, the patient is slightly drowsy, but responsive to commands as well as alert and oriented. Primary survey does not reveal any abnormalities and vitals are unremarkable. Remainder of examination reveals that the patient is unable to move her left wrist, but distal cap refill is less than 2 seconds and sensation is intact. Ecchymosis is present on the patient's left forehead. Past history is significant for high blood pressure, diabetes and atrial fibrillation for which the patient takes coumadin, atenolol and metformin. X-ray of the left wrist, chest X-ray (CXR) and computed tomography (CT) head are ordered. Patient is found to have both a Colle's fracture and small subdural hematoma in the left frontal lobe. Fresh frozen plasma (FFP) is ordered and neurosurgical consultation is obtained. What other steps in management should be taken? What special considerations should be taken into account in the elderly population?

DISCUSSION

Overview

- Less likely to be injured than younger individuals
- More likely to have fatality
- Possible for 80% to return to pre-existing state

- Must take pre-existing disease into account.
- Leading causes of death from injury:
  - *Falls*: Most common cause of unintentional injury. Keep in mind that minor mechanism of injury may cause serious harm especially when anticoagulation is on board.
  - *Motor Vehicle Accidents*: Be aware that as patients age, they have worsening daylight acuity, glare resistance and night vision.
  - *Burns*: Be aware of limited mobility. They may worsen burn risk and severity of burns.

Airway

- Elderly patients have limited cardiopulmonary reserve and therefore the threshold for intubation should be lower.
- Considerations:
  - Dentition
  - Nasopharyngeal fragility
  - Macroglossia
  - Microstomia
  - *Cervical arthritis*: Higher risk for cord injury from undue manipulation.

Breathing and Ventilation

- Supplemental oxygen placement; be careful with chronic CO_2 retainers.
- Lifelong exposure to environmental toxins and possibly tobacco smoke.
- Chest injuries have higher mortality rate.
- Rib fractures and pulmonary contusions are common, but poorly tolerated.

Circulation

- Progressive loss of cardiac function with aging.
- Predisposition to re-entry dysrhythmias, diastolic dysfunction from myocardial stiffness.
- Reduced creatinine clearance makes elderly patients more susceptible to kidney injury from hypovolemia, medications and other nephrotoxins.

Evaluation and Management

- Do not assume "normal" vital signs are "normal". A patient may have relatively hypotension compared to their baseline but have a "normal" blood pressure.
- Onset of abnormal vital signs such as hypotension may be delayed.

- Patients with hypotension and metabolic acidosis have very high mortality rate, especially in the setting of brain injury.
- Optimal hemoglobin level is controversial, generally higher than 10 g/dL is accepted for patients who are more than 65 years old.
- Important to identify any possible sites of bleeding early using focused abdominal sonography for trauma (FAST) or diagnostic peritoneal lavage (DPL) due to limited cardiopulmonary reserve.
- Be careful not to miss bleeding in the retroperitoneum.

Disability

- Brain and spinal cord injuries
- Decreased brain weight with replacement by cerebrospinal fluid (CSF) and more tightly adhered dura; decreased risk of contusion, but increased risk of parasagittal bridging vein rupture due to stretching. Also allows more blood to be collected before symptoms are apparent.
- Loss of water and protein making the intervertebral disks more compressible. Higher risk of spine and spinal cord injury.
- Osteoarthritis causes canal stenosis, segmental instability, and kyphotic deformity may complicate management; increased risk of central cord compression. It may need magnetic resonance imaging (MRI) evaluation; makes fracture diagnosis more difficult.
- Much higher risk of subdural hematomas.

Exposure and Environment

- Consider that elderly individuals have decreased ability to regulate their thermal temperature.
- Decrease dermal barrier against bacteria.
- Impaired wound healing.

Musculoskeletal

- Increased risk of fracture to long bones.
- Most common areas of fracture are proximal femur, hip, humerus and wrist.

SPECIAL CONSIDERATIONS[1]

- Drug interactions
- Beta blockade may cause peripheral vasoconstriction and hypotension.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) or anticoagulants may contribute to blood loss.

- Steroids may reduce immune response.
- Diuretics may lead to dehydration.

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1. Alexander BH, Rivara FP, Wolf ME. The cost and frequency of hospitalizatoin for fall-related injuries in older adults. Am J Public Health. 1992;82:1020-3.

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2. Oreskovich MR, Howard JD, Copass MK, et al. Geriatric trauma: Injury patterns and outcome. J Trauma. 1984;24:565-72.

Case Study 89: Pelvic Fracture

CASE HISTORY

A 31-year-old male is presented to the trauma bay after a motor vehicle accident (MVA). The patient was a restrained driver when his vehicle was T-boned on the driver's side by another car. When patient arrives by emergency medical services (EMS), he is alert and oriented to person, place and time with cervical-spine collar in place. Vitals are temperature 37.5°C, blood pressure (BP) 100/70 mm Hg, pulse rate 110 beats/minute, respiratory rate 25 breaths/minute and O_2 saturation level of 98% on room air. Primary survey reveals intact airway, breathing and circulation. On secondary survey, patient has tenderness on his right hip, with evidence of bony instability when anterior pressure is applied. A right lateral compression fracture is suspected. Hip binder is placed, a bolus of normal saline is started and patient is sent for further imaging. What imaging should be ordered? What should be considered in the management of pelvic fractures?

APPROACH TO PELVIC FRACTURES

When evaluating pelvic fractures, an understanding of the mechanism of injury is very important. The damage done during a small fall versus a MVA at 90 mph will be very different. Symptoms of pelvic injuries are often nonspecific and any pain from the mid-thigh to the mid-abdomen should raise concern. Due to the risk of massive blood loss with pelvic injuries, it is very important that the pelvis is never rocked during the physical examination. Every time the pelvis is rocked the patient may lose upto 2 units of blood. When examining the pelvis, grab the iliac crests and push in. Any movement suggests bony instability. If the patient is awake and alert with no pain or tenderness on examination, a pelvic X-ray is not necessary. The gold standard for viewing a pelvic fracture is a 3D reconstructed computed tomography (CT) scan which gives an excellent understanding and can also diagnose the presence of retroperitoneal hemorrhage.

CLASSIFICATIONS OF PELVIC FRACTURES

Lateral Compression Pelvic Fractures

These are caused by lateral impact from a T-bone MVA. The impact causes pelvis to shorten and implode, and the force is applied through back of sacrum so the ligaments stay intact. These generally do not bleed,

therefore if a patient has hypotension with a lateral compression pelvic fracture you should search elsewhere for an intra-abdominal bleed or blunt aortic injury (Figs 1A to C).

Anterior Pressure Compression Fracture

These are caused by a head-on MVA or head-on blunt force such as during horseback riding causing the pelvis to explode. There is pure ligamentous rupture resulting in lots of bleeding requiring an average transfusion of 5–6 units in 24 hours (Figs 2 and 3).

Vertical Shear

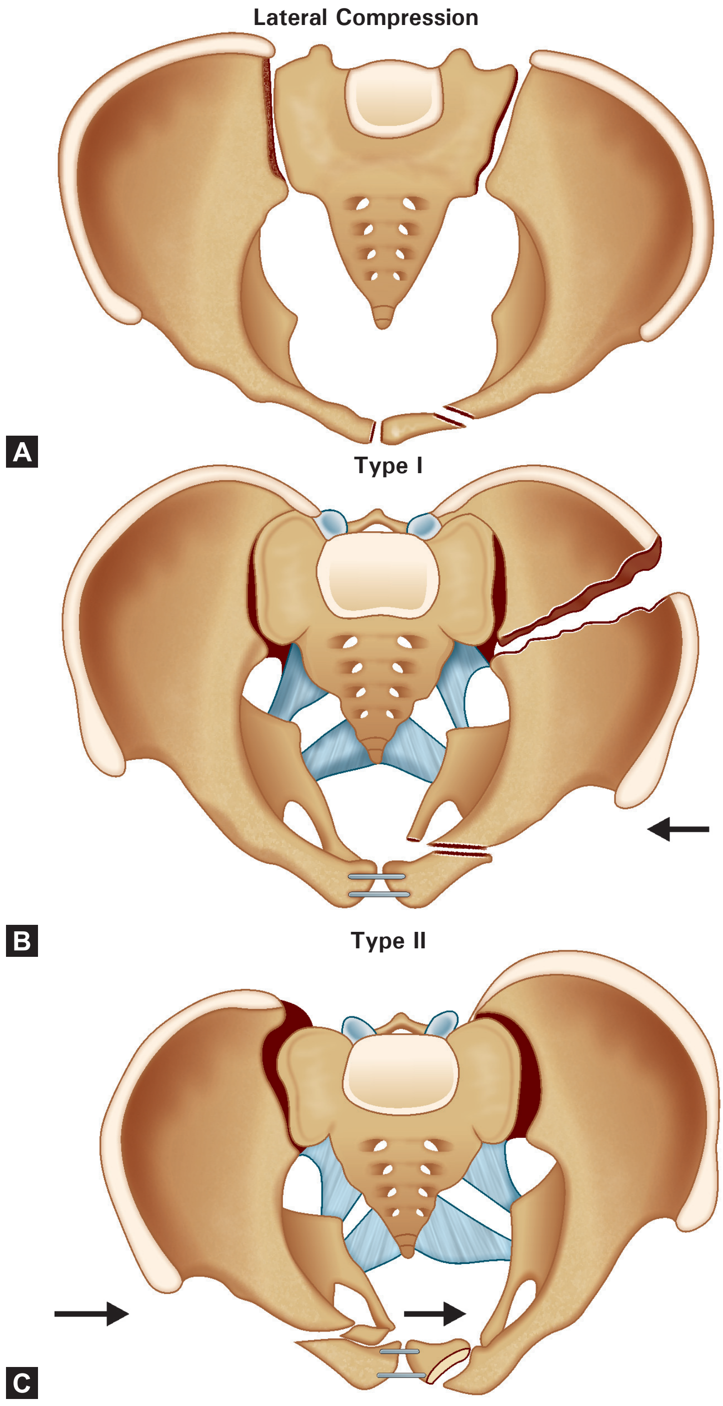
These result from a fall from a height or head-on motorcycle accident with legs outstretched and are often visually impressive. The diagnosis can be made on physical examination, if one iliac crest is higher than the other. These fractures usually have minimal bleeding.

TREATMENT OF PELVIC FRACTURES

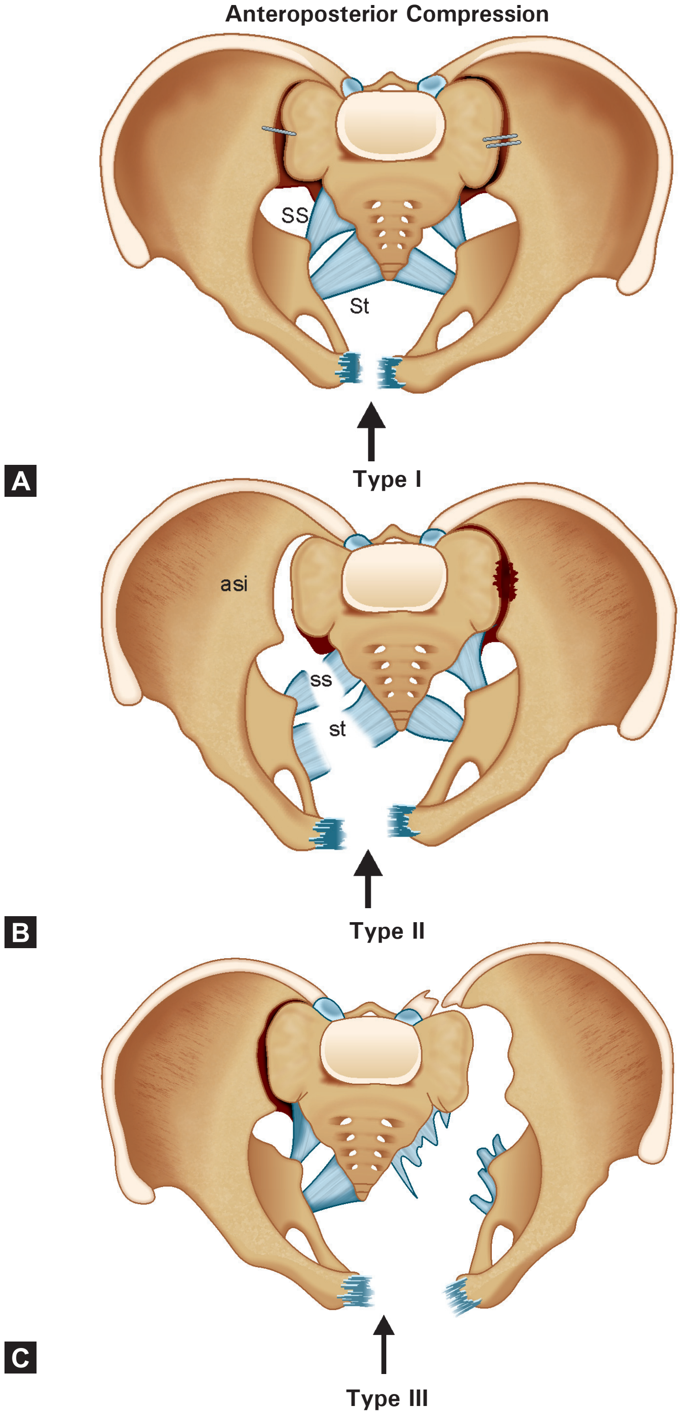
Any patient with a significant pelvic fracture should be intubated because they may decompensate quickly. You should also have blood ready for transfusion as significant bleeding is often present. A brief lower extremity neurologic assessment should be performed in order to obtain a baseline and assessment of the injury. Other sites of bleeding, such as the abdomen and thorax, should also be excluded. The best method of resuscitation of a patient with a pelvic fracture is giving blood, platelets and fresh frozen plasma early, and later lactated ringer's solution and normal saline. Factor VII can also be used to help normalize the international normalized ratio and stop the bleeding. The dose should be 100 mcg/kg of body weight for severe bleeding.

Open pelvic fractures can cause exsanguinating hemorrhage. To stop the bleeding, pack the hole and push hard. It is okay to make the opening bigger to get the packing in. Vicryl mesh and fibrin glue can also be used followed by the towels and gauze. Angiography is required early for open fractures but laparotomy is not required early on. Immediately call for operating room and orthopedics simultaneously (or transfer if needed). Do not explore the wound in emergency department.

Bleeding can not necessarily be predicted based on fracture pattern because lots of low-grade injuries and lateral compression fractures do bleed. The low-grade injuries do suggest that you should look elsewhere, but you must be aware that the fracture is also a possible source of the bleeding. It is especially important to be aware that, in older adults, lateral compression fractures often bleed and are highly lethal. At some point everyone with a pelvic fracture needs an abdominal CT even with a negative focused assessment with sonography for trauma (FAST) scan.



Figs 1A to C: (A) Sacral compression Fx on side of impact; (B) Iliac wing fracture on side of impact; (C) LC type I or II fracture + contralateral Anteroposterior compression fracture.



Figs 2A to C: (A) Pubic rami or ligament disruption; Slight widening of symphysis; (B) Iliac wings rotated externally "hinging" at SI joint posterior aspect; Open book; (C) Complete disruption of sacroiliac ligaments; unstable.

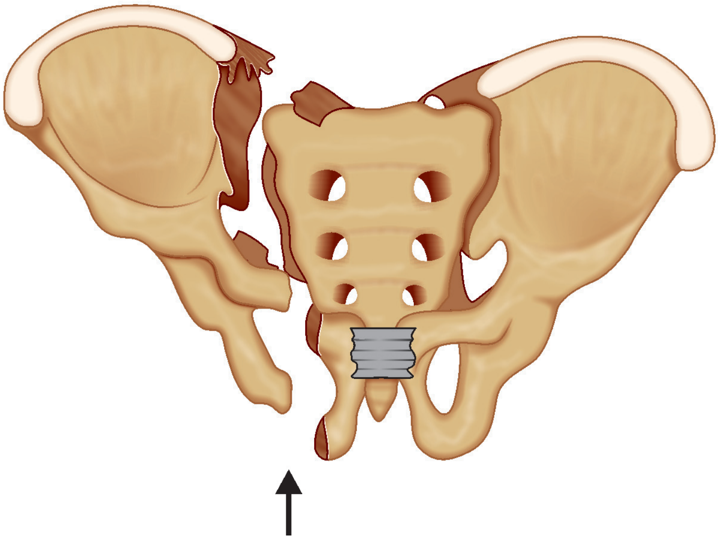


Fig. 3: Full from heights;
Anterior: Both public ramis fratured; *Posterior*: SI complex or sacral Fracture

Controlling Pelvic Hemorrhage

The goal for controlling pelvic hemorrhage is stabilizing the clot via external compression and reduction of the pelvic fragments. This can be done through any of the following methods:

Bed sheet: Place a bed sheet on a gurney, place the patient on the sheet, Bedsheet to control the sheet and crisscross and tie the sheet over the patient (Fig. 4).

Medical anti-shock trousers: The method uses a pelvic splint which provides pressure. If the abdominal part is used, you must also use lower extremity part as well. This raises the BP by increasing systemic vascular resistance. The major complication is that it also increases abdominal pressure which may precipitate abdominal compartment syndrome. However, it is still very effective at treating bleeding.

External fixation: This can be rapidly applied and may be definitive. It can also disrupt the view on CT and may increase displacement of certain elements.

Binder: These are placed on the trochanters. It is a Velcro apparatus that makes everything simpler and is very effective to the point that it will fix the displacement.

Angiography: This is an excellent test to achieve hemostasis and is both diagnostic and therapeutic; however, it does take time to set up. The use of angiography varies by institution so it helps to have an algorithm in place for indications at your institution.

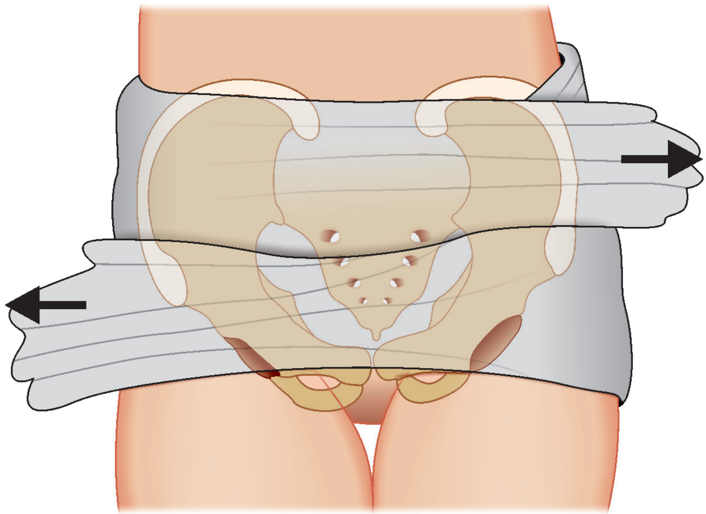


Fig. 4: Bedsheet/Binder used to control bleeding

COMMON ACCOMPANYING INJURIES

Pelvic fractures frequently occur along with other injuries due to the severity of the mechanisms of injury that cause pelvic fractures. Urethral injuries are common comorbidities. These require a retrograde urethrogram for evaluation before a Foley catheter can be safely inserted. If the urethrogram shows a urethral injury that contraindicated the use of a Foley catheter, a suprapubic tube should be used. Bladder ruptures can also be seen with pelvic fractures and usually indicated by gross hematuria. A CT cystogram is diagnostic.

OUTCOME

The morbidity of pelvic fractures is often high but mortality is low due to the institution of good multidisciplinary approaches. However, these results are time dependent. All aspects must be managed simultaneously, not by a step-by-step approach.

ADDITIONAL READING

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2. Journal of TRAUMA® Injury, Infection, and Critical Care • Volume 71, Number 6, December 2011 EAST Pelvic Fracture Guidelines.

Case Study 90: Compartment Syndrome

CASE HISTORY

A 32-year-old man's right leg is trapped beneath his overturned car for nearly 2 hours before he is extricated. On arrival in the emergency room, his right lower leg is cool, mottled, insensate and motionless. Despite normal vitals signs, pulses cannot be palpated below the femoral vessel and the muscles of the lower extremity are firm and hard. During the initial assessment of this patient, which of the following is most likely to improve the chances for limb salvage?

DISCUSSION

Compartment syndrome causes generalized painful swelling and increases pressure which deprives the muscle and nerves of oxygen and nutrients. This may lead to neurologic deficit, muscle necrosis, ischemic contracture, infection, delayed healing and possible amputation. Common sites for compartment syndrome include: lower leg, forearm, foot, gluteal region and thigh. Injuries such as tibial and forearm fracture, crush injury to muscle, burns, excessive exercise, prolonged external pressure to an extremity, and use of extremity immobilization devices, such as dressing or casts, may increase the risk for compartment syndrome. Early diagnosis is essential as a treatment.

SIGNS AND SYMPTOMS

These include increased or severe pain, asymmetry, palpable tenderness, altered sensation, weak or nonpalpable pulses and pain on passive stretch. Pressure measurement greater than 30–45 mm Hg indicates decreased blood flow, which may result in muscle damage.

MANAGEMENT

Fasciotomy is a surgical procedure where the fascia is cut to relieve tension or pressure. Compartment syndrome is one of the conditions where the fasciotomy is indicated. Patient is monitored for 30 minutes and if no change is observed in that time period then fasciotomy is urgent (Fig. 1). Delay in the treatment may result in myoglobinuria and as a result decrease in renal function occurs.

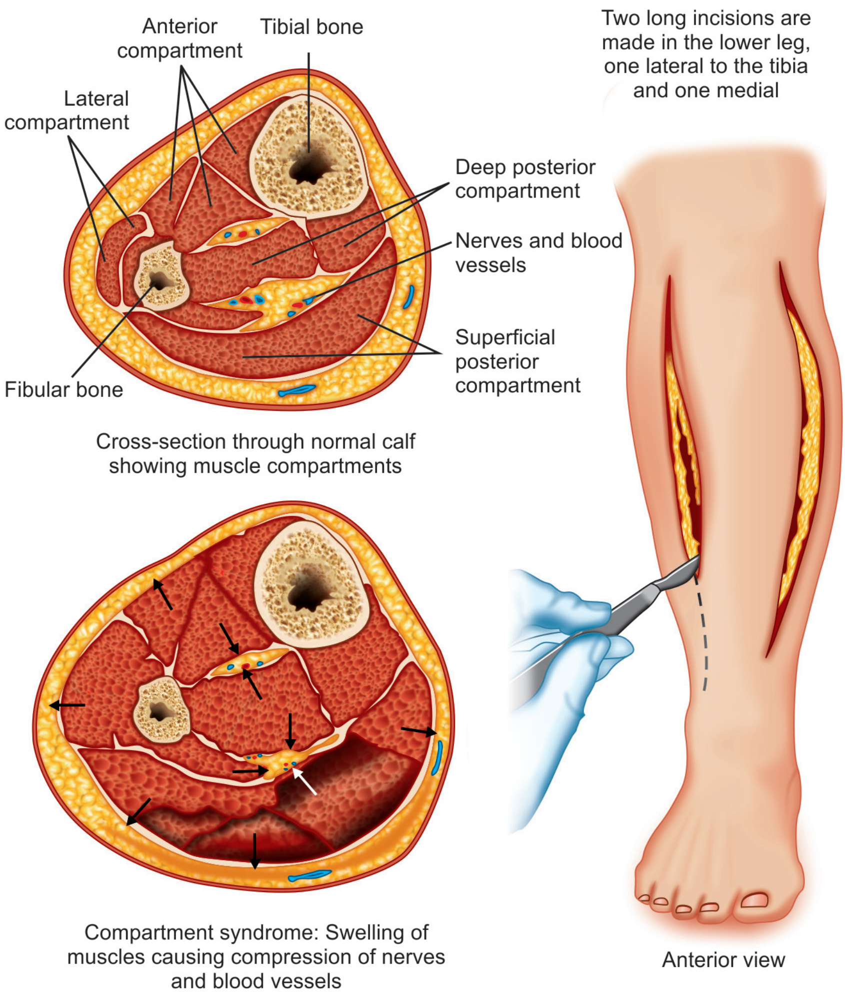

Fig. 1: Compartment syndrome with fasciotomy procedure

Source: Nucleus Medical Media. Compartment Syndrome with Fasciotomy Procedure — Medical Illustration, Human Anatomy Drawing. [online] Available from catalog.nucleusinc.com/generateexhibit.php?ID=173. [Accessed September, 2012].

CHAPTER

16

Infectious Diseases

Case Study 91: Meningococcemia

CASE HISTORY

A 19-year-old college student who lives in a dormitory is presented to the emergency department (ED) with the chief complaint of fever, chills, rigor, throat pain, joint pain and muscle aches. He denies foreign travel. He presents with maculopapular rash, petechiae and purpuric spots. Upon examination, the patient has a temperature of 104°F (40°C). A blood culture, complete blood count (CBC) and skin biopsy are ordered by the ED doctor with the assumption that the rash may be meningococcemia.

DIFFERENTIAL DIAGNOSIS

Any ill appearing or toxic looking patient with a petechial rash and associated symptoms ranging from pharyngitis to meningitis to bacteremia will make you think this is a potentially fatal infectious disease. The differential diagnosis includes:

- Rocky Mountain spotted fever
- Gonococcemia
- Bacterial endocarditis
- Toxic Shock Syndrome (TSS)
- Vasculitis
- Disseminated Intravascular Coagulation (DIC)

DISCUSSION

Meningococcemia is caused by *Neisseria meningitidis* in the blood leading to a wide range of symptoms (Figs 1 to 4). Patients present with headache, decreased sensorium, neck rigidity, rash and hypotension. If meningococcemia is suspected, treatment should be started promptly.[1] Early treatment usually results in good outcomes. If the patient is in shock, has disseminated intravascular coagulation (DIC) or kidney failure, the prognosis is not as positive.

Fig. 1: Meningococcemia
Courtesy: Logical Images, Inc. 2009.

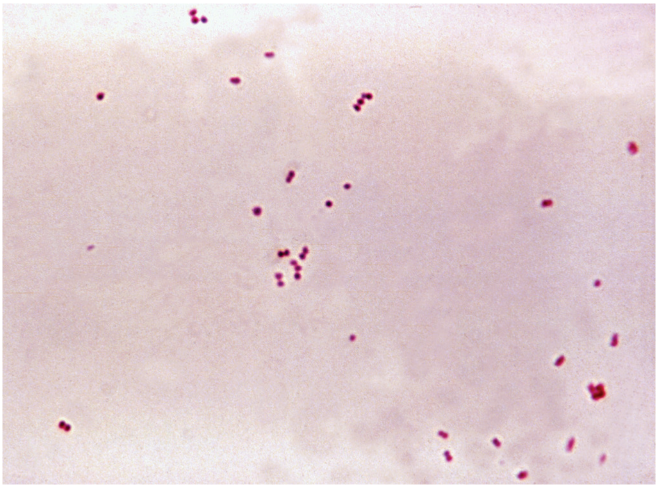

Fig. 2: Gram-negative *Neisseria meningitidis* diplococcal bacteria, 1150X
Source: Available online at http://www.cdc.gov/meningococcal/index.html

Meningococcal disease[2] is important to recognize and treat quickly as severe complications can rapidly arise, as in the high-profile 2004 case of "Miracle Baby Charlotte" (Fig. 5). Within 30 minutes of the first appearance of a petechial rash, her entire body was swollen, purple, and her limbs were blackening. She required resuscitation twice in the first day and ultimately all four limbs were amputated as a result of gangrene. Her parents decided to use her publicity to raise awareness about the speed of this disease and to encourage research of the development of vaccines.

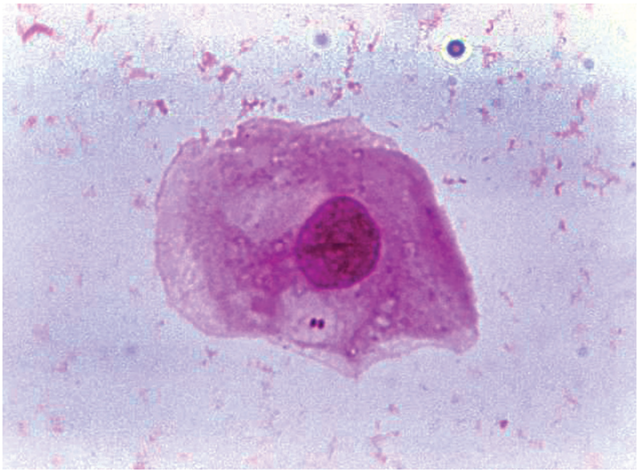

Fig. 3: *Neisseria meningitidis*; photomicrograph, 1125X
Source: Available online at http://www.cdc.gov/meningococcal/index.html

Fig. 4: A 4-month-old girl with gangrene of hands secondary to meningococcemia-induced arterial occlusions
Source: Available online at http://www.cdc.gov/meningococcal/index.html

When dealing with patients with meningococcemia, 24 hours of airborne isolation is often ordered until the patient is stable and some antibiotics have been able to work in their system. Dexamethasone and antibiotics are often given intravenously. The addition of dexamethasone as an adjunct in bacterial meningitis cases has shown effectiveness in reducing the mortality rate and hearing loss for patients in developing countries.[1] The same benefits have been seen less in populations heavily affected by human immunodeficiency virus (HIV). The patient may also receive fluids, treatment for hypotension or other symptoms associated with the disease.

Fig. 5: "Miracle Baby" Charlotte Cleverley-Bisman, with peripheral complications of meningococcal disease
Courtesy: wikimedia.org

Emergency Department Treatment and Disposition:

- Give Ceftriaxone 2g IV and Vancomycin 1g IV empirically pending blood cultures/CSF cultures and skin culture.
- Patient needs to be hospitalized under infectious disease consult for anticipated complications like Fredrickson House Syndrome.

PROPHYLAXIS, PREVENTION AND TREATMENT OF CONTACTS

The most effective way to prevent this dreadful, fatal disease is to complete the recommended vaccine schedule. 24 hours isolation followed by treating the contacts with Prophylactic antibiotics.

If one member of a household has the disease, prophylactic antibiotics are often given to the rest of the family for prevention purposes. Also, a vaccine exists that protects against some variations of meningococcus (not all). This vaccine is recommended for children and anyone moving into a dormitory. The vaccine is often given 3 weeks before the student leaves for college.

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CHAPTER

17

Renal Emergencies

Case Study 92: Nephrolithiasis/Renal Colic

CASE HISTORY

A 45-year-old male presents with flank abdominal pain for 1 day. Pain started suddenly in his right side and then began to radiate to his right groin. Pain is sharp, 9/10, and waxes and wanes. No nausea, vomiting, or diarrhea is noticed. Patient denies having a pain like this in the past or ever having kidney stones. He does report that he thinks his father did have a stone at one time. Patient denies any abdominal surgeries including appendectomy. He has no known medical problems. On physical examination, vitals are significant for mildly elevated pulse and blood pressure. On further examination, patient is nontender with normal external genital examination. Computed tomography (CT) of abdomen/pelvis is ordered and is found to have evidence of a stone in the right ureter. No evidence of hydronephrosis or other obstructing signs are found. Patient is discharged with pain medication with instructions to increase fluid intake and follow-up with *primary care physician* (PCP).

DISCUSSION

Patient is a male presenting with right-sided abdominal pain. Differential diagnosis includes appendicitis, testicular torsion, hernia and nephrolithiasis. Physical examination did not reveal any signs of torsion or hernia and therefore CT was done to further rule out appendicitis and nephrolithiasis. Patient had evidence of a small stone that would most likely pass on its own, and therefore no further intervention was needed.

Ureterolithiasis Discussion

Tamsulosin for Ureteral Stones in the Emergency Department: A Randomized, Controlled Trial.

- Renal colic, stones that are lodged in distal ureter.
- The main factors affecting the retention of ureteral calculi are:
    - Ureteral muscle spasm
    - Submucosal edema
    - Pain and infection
- α1 receptors are predominant in the ureteral smooth muscle.
- Blockade of these α-adrenergic receptors would decrease ureteral peristaltic amplitude and frequency.
- Decreasing intraureteral pressure and allowing increased fluid transport to occur.

Tamsulosin:
- Selective α1A and α1D adrenoreceptor blocker.
- Initial treatment of patients with lower urinary tract symptoms such as benign prostatic hyperplasia (BPH).
- Use of tamsulosin or other selective adrenoreceptor blockers + standardized pain control regimen in patients with distal ureterolithiasis.

Interventions (Fig. 1)
- The treatment group received tamsulosin hydrochloride 0.4 mg by mouth daily for 10 days + standard analgesic therapy.
- All subjects also received standard discharge instructions for renal colic and were given a urine strainer and instructions on straining their urine and collecting debris.
- All patients were instructed to follow-up with the hospital's on-call urologist in 10–14 days.

Primary outcome:
- Successful spontaneous ureteral stone expulsion at 14 days (Fig. 2).

Secondary outcomes: Below mentioned are the secondary outcomes.
- Time to stone passage
- Self-reported pain scores
- Number of colicky pain episodes
- Number of unscheduled return to ED/primary care visits
- Number of days of missed work/usual function
- Amount of analgesic used
- Adverse events
- All outcomes were evaluated at the 2, 5 and 14-day telephone follow-up sessions for patient information (Flow chart 1).

See related article on preventing kidney stones.

Stay Hydrated

Staying hydrated is not as simple as just drinking water. Other things to consider include:
- Do not overdo it. Avoid drinking more than eight 8-ounce glasses of water a day. More water than this can change the balance of particles

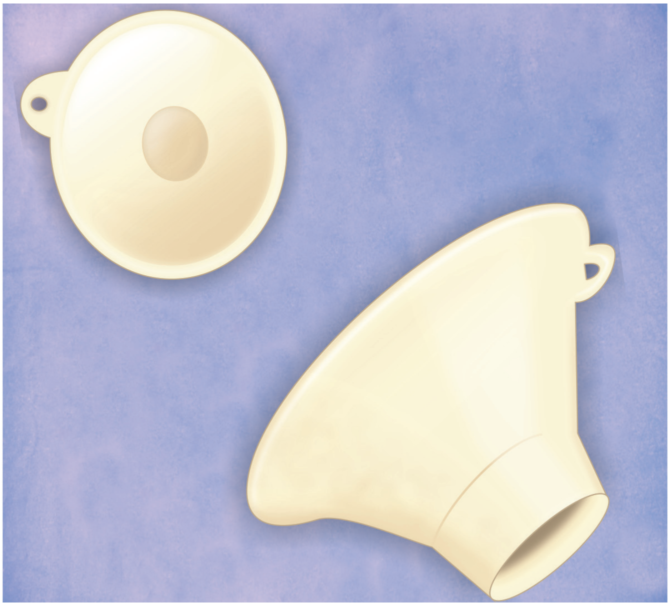


Fig. 1: Sieve used to check urine for passed stones

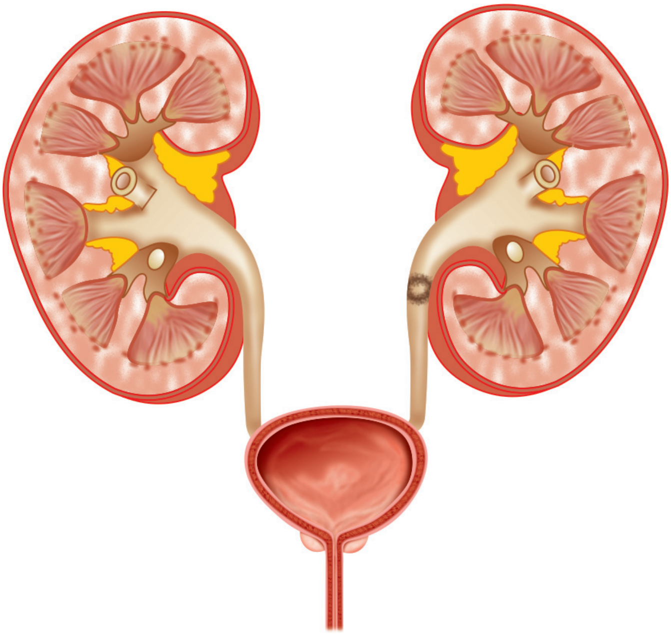
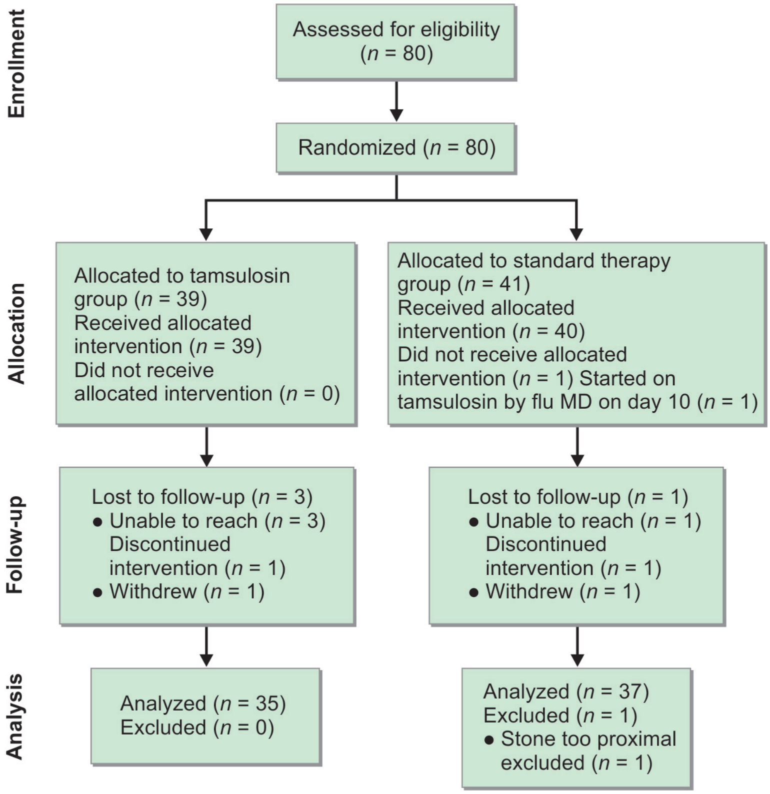


Fig. 2: Successful spontaneous ureteral stone expulsion

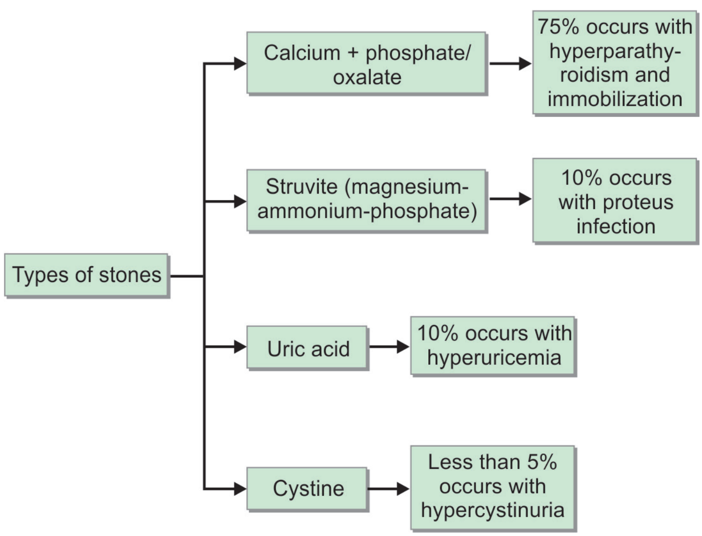
in your body called electrolytes. This can be harmful and sometimes happens in endurance athletes, such as marathon runners, who drink too much water when losing a lot of sweat. In such circumstances, a

Flow chart 1: Follow up case study with tamsulosin



Source: Preventing kidney stones with diet and nutrition. Am Fam Physician. 2011; 84(11):1243-4.

Flow chart 2: Types of stones



Source: Badar M Zaheer MD

Fig. 3: These spiked rods are uric acid crystals photographed under polarized light. Increased uric acid blood levels and formation of uric acid crystals in the joints are associated with gout

Courtesy: A.D.A.M. Health Solutions, Ebix, Inc. (2012). Uric acid crystals. [online] Available from www.nlm.nih.gov/medlineplus/ency/imagepages/1222.htm. [Accessed August, 2012].

mixture of water, electrolytes and a small amount of sugar can be used. Examples are chicken broth, coconut water, pedialyte, or use of oral rehydration salts. Artificial sweeteners should be avoided because they have the opposite effect, making it more difficult to rehydrate.

- Avoid sugary drinks, such as fruit drinks and sports drinks, because they add calories and change the acid-base balance of the urine.
- For most kidney stones (Flow chart 2 and Fig. 3), urine should be less acidic. One way to make the urine less acidic is to add citrate to drinking water. Lemon and lime juices are great sources of citrate.
- You can also breathe in moisture to stay hydrated by using humidifiers and steam.
- Be aware that obesity increases the risk of dehydration. The more extra weight someone carries, the more important hydration becomes.

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CHAPTER

18

Medical Errors

Case Study 93: Laboratory Error

"More people are killed each year by medical errors than by traffic accidents"
Source: NHTSA.gov

—*Badar M Zaheer*

INTRODUCTION

The following case presentation is of a patient who was initially presented with profound electrolyte abnormalities that were later found to be laboratory error.

Patient safety can be greatly influenced by the presence of laboratory errors. There is a large focus on improving patient safety in the healthcare field right now including analyzing various performance measures to determine how to meet higher standards in patient safety.[1]

CASE HISTORY

A 64-year-old man is presented to the emergency department (ED) at 1630 hours with 3 days of abdominal pain and 1 day of vomiting. He had also developed significant pallor and diaphoresis.

His past medical history included Type 2 diabetes mellitus, hypertension (HTN), and dyslipidemia. He had no history of tobacco, alcohol, or recreational drug use. His medications included glipizide 10 mg twice a day (bid), Lipitor® 20 mg daily, cyclobenzaprine three times a day as often as needed (tid prn), metformin 1,000 mg bid, lisinopril 40 mg bid, and Coreg 12.5 mg bid.

On examination, he was found to be hypotensive with blood pressure of 71/42 mm Hg and pulse rate of 84 beats/minute, respiratory rate 22 breaths/minute, temperature 98.3°F, and oxygen saturation 98% on room air. Physical examination showed lethargic patient in moderate to severe distress. Skin examination was positive for pallor and diaphoresis, and abdominal examination was consistent with distension and ascites. Musculoskeletal examination showed an erythematous and ulcerated left

great toe with yellow drainage. The remainder of the physical examination was within normal limits.

The patient was given a 1 L bolus of 0.9% normal saline starting at 1635 hours and ending at 1745 hours. Complete blood count (CBC) and basic metabolic panel (BMP) were drawn and showed numerous critical values. Although he did not look severely anemic, his hemoglobin level was 4.8 g/dL, had a hematocrit value of 14.2%,and thrombocytopenic with a platelet count of 45 × 103/μL, as per labs which is unbelievable. The most shocking findings were his comprehensive metabolic panel (CMP) significant for potassium of 1.7 mEq/L chloride of 127 mEq/L, bicarbonate of 7 mEq/L, blood urea nitrogen (BUN) of 36.9 mg/dL, creatinine of 3.0 mg/ dL, anion gap of 8, calcium of 3 mg/dL, magnesium of 0.6 mg/dL and glucose of 150 mg/dL. Blood pressure had improved to 100/54 mm Hg after a normal saline bolus. Electrocardiogram was done at 17:22 hours and showed normal sinus rhythm.

Due to the concern for sepsis, the patient was given intravenous (IV) Rocephin 2 gm at 17:30 hours. A second bolus of 0.9% normal saline was started at 17:45 hours along with IV flagyl 500 mg at 17:50 hours and 20 mEq/L of potassium chloride (KCl) IV at 17:55 hours for potassium replacement. The patient was transferred by medical force to another institution at 18:25 hours.

On arrival to the second institution, laboratory tests were repeated. CBC showed a hemoglobin level of 12.2 g/dL, hematocrit value of 33.5% and reticulocyte count of 0.6%. CMP showed a chloride of 99 mEq/L, potassium of 4.7 mEq/L, bicarbonate of 20 mEq/L, BUN of 89 mg/dL, creatinine of 2.49 mg/dL, calcium of 7.6 mg/dL, magnesium of 1.6 mg/dL, glucose of 330 mg/dL and lactic acid of 1.6 mg/dL.

DISCUSSION

The patient in this case is presented to the ED in shock and was subsequently found to have several severe electrolyte abnormalities. After transfer to an outside institution, his laboratory tests were found to be much closer to normal limits. The patient was only given 20 mEq/L of KCl for replacement and potassium was found to have risen from 1.7 mEq/L to 4.7 mEq/L. There should not have been a rise of that magnitude from that amount of KCl replacement. Additionally, his magnesium was found to have risen from 0.6 mg/dL to 1.6 mg/dL, although no replacement doses had been given. It is clear that the first set of results were likely due to laboratory error.

The lesson to be learned from this case is the importance of being aware of the possibility of laboratory error. In this particular patient, all of

Table 1: Confused drugs and results of error

Confused drugs	*Results of error*
Novolin, Novolog, and Novolin 70/30	Hypoglycemia and poor control of diabetes
Clonidine vs Klonopin	Hypotension, loss of seizure control
Ambisome, Abelcet,Amphocine (Fungizone)	Respiratory arrest, renal failure
Metformin vs Metronidazole	Hypoglycemia or untreated infection
Vinblastine vs Vincristine	Due to differences in dosages this error has resulted in fatalities
Tramadol, trazodone, toradol	Failure of pain control, change in psychiatric state
Coumadin, Avandia, Cardura	Coagulation complications
Hydromorphone vs morphine	Opioid overdose due to hydromorphone's higher potency
Celebrex, Cerebyx, Celexa	Alterations in mental status, failure of pain control, failure of seizure control

Source: Dr Badar M Zaheer

the measured values were significantly abnormal, making it more likely to be a laboratory error. Additionally, this patient is presented with no symptoms and no electrocardiography (ECG) evidence of hypokalemia or hypomagnesemia, yet laboratory results showed a profound deficiency.[2] This would be very unlikely.

Being aware of these potential errors can be life saving to patients. This patient was given 20 mEq/L of KCl before arriving at the transferred institution. Had this patient been given additional doses of potassium he could have been at risk for the effects of hyperkalemia including torsades de pointes and cardiac arrest.[3] Additionally, on repeating magnesium measurement it was found to be near normal. Had this patient been given magnesium replacement initially, he could have been at risk for cardiotoxic effects.[4] Also, imagine if we had given blood transfusion, depending on the lab findings. It could have been a serious health risk and caused unnecessary complications from transfusion.

With 1-in-3 hospital patients accidentally harmed every year in the United States hospitals (Campaign Zero), it is important to find any ways possible to reduce errors. Using a "cognitive break" during diagnosis can help prevent errors (Table 1). This means to take a break from mental, visual and auditory stimulation for 1–2 minutes and then think clearly before making a decision.

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CHAPTER

19 Water and Electrolyte Imbalance

Case Study 94: Hypokalemia

CASE HISTORY

A 76-year-old female has been experiencing severe diarrhea and vomiting for the past 3 days. She is generally in good health otherwise with only mild hypertension and hyperlipidemia, which are well controlled with hydrochlorothiazide (HCTZ) and atorvastatin, respectively. After 2 days of vomiting and diarrhea, she began feeling fatigued with muscle weakness and soreness. On the 3rd day, she became more weak and felt multiple palpitations in Figure 1. She called emergency services and was transported to the emergency department promptly. Upon arrival, she appeared hypovolemic and dehydrated with pale conjunctiva and prominent skin turgor abnormality. She was started on intravenous (IV) normal saline, an electrocardiogram (ECG) was performed, and routine labs were drawn. The ECG showed flat T-wave with an additional small wave following it and occasional premature ventricular contractions (PVCs). Her labs returned with a serum potassium level of 2.4 mEq/L. IV normal saline was continued, and IV K^+ was given for 2 hours. Over the next 2 days, she was given oral K^+ supplementation and IV fluid hydration. Her diarrhea and vomiting subsided, along with all of her prior symptoms. On the 3rd day of hospitalization, she was discharged and her HCTZ was restarted.

BRIEF CASE DISCUSSION

This patient developed hypokalemia due to several mechanisms: (1) diarrhea and vomiting, and (2) HCTZ diuretic use. The combination of the two makes it more likely to result in significant hypokalemia. Potassium is very important to the impulses necessary for proper muscle contraction. Therefore, abnormalities in potassium levels may lead to various muscle-related problems, including weakness, soreness and palpitations. She presented with the classic ECG finding of flattened

Fig. 1: 7 Ls to remember: Symptoms of hypokalemia
Source: Dr Badar M Zaheer

T-wave as well as a U-wave, see figure 2. Since she was presenting with severely low potassium and cardiac arrhythmias (i.e. PVCs), potassium repletion was initially performed via IV infusion and then was switched to oral. If the vomiting and diarrhea remained intractable, IV supplementation could have been continued until the vomiting and diarrhea subsides.

HYPOKALEMIA DISCUSSION

General

Serum potassium levels are normally in the range of 3.5–5.0 mEq/L and therefore hypokalemia is defined as a concentration of less than 3.5 mEq/L (*Source*: 5MinuteConsult). Although hypokalemia can occur in any individual, the frequency increases with age, likely due to a higher use of diuretics and a poor diet (*Source*: Medscape). Furthermore, there are suggestions that hypokalemia is more prevalent in African

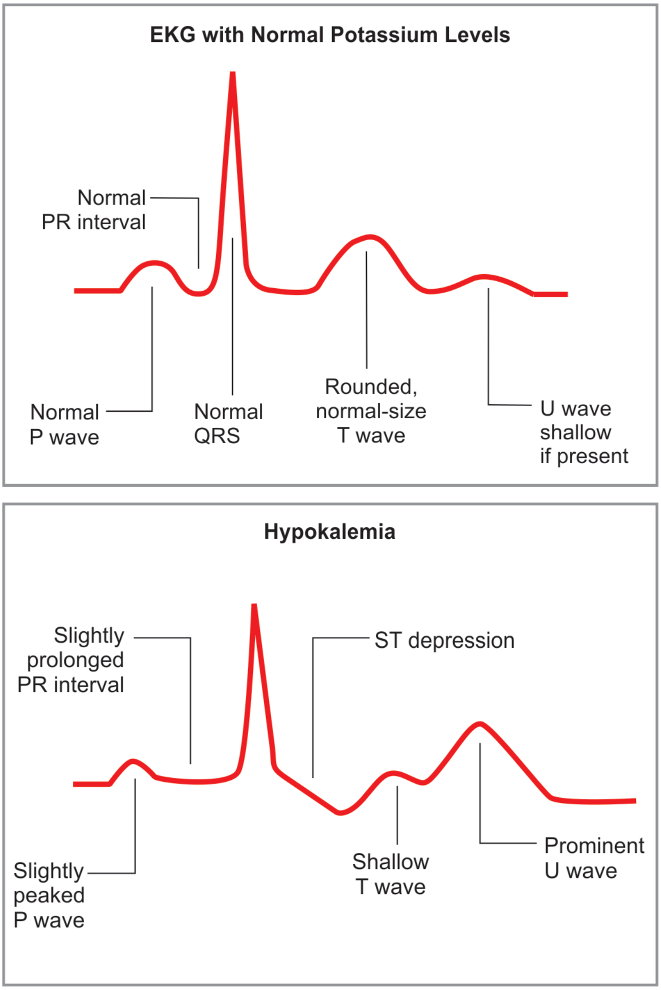

Fig. 2: EKG changes
Source: Dr Badar M Zaheer

Americans and women. High risk groups include person with eating disorder, alcoholism, acquired immunodeficiency syndrome (AIDS), and those who have had bariatric surgery.

Pathophysiology and Causes

There are three mechanisms that can result in hypokalemia (*Source*: Medscape and 5MinuteConsult): (1) deficient intake, (2) increased excretion and (3) shift from extracellular to intracellular space. Isolated dietary deficiency is relatively uncommon, but can occur in anorexia nervosa, alcoholism, elderly people with very poor dietary intake, and those with prolonged total parenteral nutrition (TPN) with

inadequate potassium supplementation. Increased excretion is the most common cause of hypokalemia overall. This can occur from increased sodium delivery to the renal collecting ducts such as with diuretics (i.e. more sodium to exchange for potassium), mineralocorticoid excess (i.e. primary or secondary hyperaldosteronism), diarrhea, vomiting, nasogastric suction, laxative abuse, bulimia, and metabolic alkalosis (i.e. fewer hydrogen ions to exchange for potassium). The final mechanism is a potassium shift from the extracellular to intracellular space, which often accompanies the increased excretion mechanism. Since the total body potassium is not changed with these potassium shifts, episodes are often transient and self-limited. These intracellular shifts can occur due to alkalosis, insulin excess (endogenous or exogenous), and beta-adrenergic stimulation (acute stress or pharmacologically induced).

Assessment

Hypokalemic patients often have no symptoms and are found incidentally with routine labs. This is generally true with mild hypokalemia in the range of 3.0–3.5 mEq/L (*Source*: 5MinuteConsult). When symptoms are present, they generally affect the variety of muscles in the body (*Source*: Medscape and 5MinuteConsult). Neuromuscular problems involve skeletal muscles and result in variable weakness ranging from mild to severe with rhabdomyolysis and/or respiratory arrest. Smooth muscle effects can cause hypomotility, constipation and ileus. Lastly, and most life-threatening, cardiac muscle effects can cause hypotension, arrhythmias and cardiac arrest. These symptoms are nonspecific and do not aid in determining the cause. For the most part, the physical examination is unrevealing as well. Muscle weakness or flaccid paralysis may be present, as well as depressed or absent reflexes. Vital signs are generally normal with occasional tachycardia or tachypnea due to muscle weakness and/or volume depletion. Although relatively nonspecific, hypertension may point toward primary hyperaldosteronism or renal artery stenosis and relative hypotension may suggest laxative use, diuretic use, excessive diarrhea and/or vomiting, or bulimia. With the symptoms and physical examination findings of moderate value in determining the cause of hypokalemia, it is important to obtain a solid patient history and to employ a diagnostic algorithm including various laboratory tests.[1] Urine tests, including potassium, sodium and osmolarity, are very important in attempting to determine the mechanism for the hypokalemia. These can all be done quickly with a spot test and the values should be analyzed together. First, a low urine potassium (< 20 mEq/L) in the setting of hypokalemia would suggest an inadequate intake, shift to intracellular space, or gastrointestinal loss, and therefore the patient should be

asked about vomiting, laxatives, diet, TPN, insulin use and bicarbonate supplements/medications. Conversely, a high urine potassium (> 40 mEq/L) suggests renal loss, in which one should evaluate the patient's medications, acid-base balance, magnesium level and blood pressure. These can help to distinguish between diuretic use, vomiting with alkalosis, mineralocorticoid excess, renal tubular acidosis and renal artery stenosis. If urine sodium is low (< 20 mEq/L) in the presence of high-urine potassium, then secondary hyperaldosteronism would be suggested. Additionally, the urine osmolarity should be considered since a highly concentrated urine (> 700 mOsm/L) will lead to a misleading absolute value of potassium and sodium. In other words, the value of the electrolyte in the urine will appear higher simply due to the fact that there is less urine volume to dilute it. Although more difficult, if one wants a more accurate measurement of urine electrolytes, a 24-hour electrolyte value can be obtained. Serum electrolyte values should be obtained as well. Low sodium, for example, may suggest the use of a thiazide diuretic as the inciting cause. A low bicarbonate level may suggest renal tubular acidosis, diarrhea, or the use of carbonic anhydrase inhibitors, while high bicarbonate level could be due to hyperaldosteronism (primary or secondary), including that due to vomiting or diuretic use. Magnesium is an important electrolyte for potassium regulation. A low magnesium can cause hypokalemia that is refractive to treatment unless the magnesium abnormality is corrected first. Most commonly hypokalemia is due to diuretics or gastrointestinal losses, but this must be confirmed. Further testing for specific causes may include serum renin, aldosterone, and cortisol, urine aldosterone and cortisol, pituitary or adrenal imaging, renal angiography, or enzyme assay for 17-beta hydroxylase deficiency. Besides determining the cause of the hypokalemia, it is also important to assess for possible severe side effects that may result. Since cardiac and skeletal muscles can be affected, an ECG and creatine kinase (CK) values should be determined. Possible ECG abnormalities may include atrial or ventricular tachyarrhythmia, flattened T-wave, or presence of a U-wave, while increased CK (creatine kinase) result in muscle breakdown or frank rhabdomyolysis.

Treatment

Treatment for hypokalemia is dependent upon the underlying cause. For all hypokalemic patients, there are some common goals: decrease further potassium losses, replenish lost potassium stores, monitor for potential toxicities, and determine the underlying cause and prevent further hypokalemic episodes. As previously stated, monitoring for toxicity includes ECG and serum CK levels. Initially, anything causing possible

potassium losses should be discontinued or addressed, including discontinuing laxative and diuretics or change to potassium-sparing diuretics, treat any diarrhea or vomiting, and control hyperglycemia. Next, potassium repletion should be done to correct the hypokalemia. As a general rule, for each 1 mEq/L decrease in potassium, there is a potassium deficit of roughly 200–400 mEq. This can either overestimate or underestimate the true potassium deficit, and therefore the serum potassium should be checked again after supplementation. The preferred form of supplementation would be an oral form of potassium since it is readily absorbed and large quantities can be given safely. If gastrointestinal upset occurs or if the patient is vomiting, an enteral form may be used. Intravenous potassium supplementation is less well-tolerated and therefore should be given much slower (< 10 mEq/hr) and monitored for local reaction due to venous irritation. Glucose supplementation should be monitored carefully since it can increase insulin production leading to more transcellular shifting of potassium into the cells resulting in lower serum potassium levels. Additionally, if the patient is acidotic, the potassium should be repleted before the acidosis is corrected. This is due to potential worsening of hypokalemia by transcellular shift as the patient becomes relatively more alkalotic. Surgical intervention is only needed if there is a specific underlying cause that can be found, such as renal artery stenosis, adrenal adenoma, or intestinal obstruction. Finally, once supplementation has returned the potassium level back to normal, it is important to monitor the potassium levels over the next few days to ensure stable values and to be sure the cause of the hypokalemia was correctly determined and treated. Ultimately, some patients may need to be on a long-term potassium supplementation and/or have long-term monitoring of serum potassium levels.

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2. Mnemonic for Hypokalemia www.allnurses.com

Case Study 95: Hyponatremia

CASE HISTORY

A 50-year-old male long-distance runner was feeling slightly fatigued when he woke up 2 days before he planned to compete in a local marathon. In an effort to help himself feel better, he began to hydrate himself by drinking large amounts of water. The following morning, he continued drinking large amounts of water since he felt that it was helping. He decided that he was well enough to compete in the marathon. During the race, he continued to drink water at every opportunity. Although he was struggling at the end, he managed to finish the race. While he was sitting in a recovery tent, he appeared slightly confused and his friends helped him hydrate with more water. This did not seem to help and over the next 15 minutes he became increasingly confused and progressively less responsive.

He was immediately transported to the emergency department. Upon arrival, he remained minimally responsive and obviously confused. Lab values were rapidly determined and showed a serum sodium (SNa) level of 124 mEq/L and a serum osmolarity of 265 mOsm/kg. On clinical examination, he did not show apparent signs of dehydration. He was immediately put on water restriction and a hypertonic saline infusion was started (Fig. 1). After 2 hours, he was no longer exhibiting any neurological deficits and his SNa was 128 mEq/L. The hypertonic saline was discontinued and over the next 48 hours he continued with water restriction and replacement of each 1 mL of urine output with 0.5 mL

Fig. 1: Hyponatremia is a risk for those exercising in excessive heat
Source: Available online at http://www.cdc.gov/meningococcal/index.html

normal saline. He continued to have routine SNa values checked every 6 hours. After 48 hours, his SNa returned to 138 mEq/L. He was discharged with no residual neurological deficits.

BRIEF CASE DISCUSSION

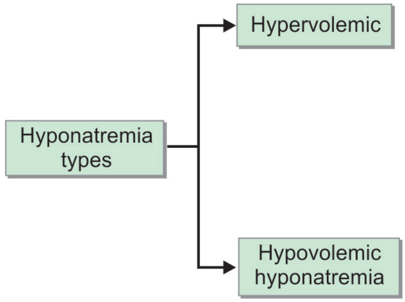
This patient developed an acute hyponatremic episode, which is considered less than 48 hours. The result of the relatively quick decrease in extracellular SNa is a fluid shift to the intracellular space that overwhelms the bodies' compensatory responses. The effects are most pronounced in the central nervous system since the cranium is a rigid structure that does not allow for expansion. The brain cells swell and cause neurological deficits as seen in this patient (e.g. confusion and decreased responsiveness). He was predisposed to hyponatremia due to several factors (Flow chart 1). These included the general stress of his illness, excessive hydration with hypotonic fluids (i.e. water), and the large amount of fluid loss (i.e. sweating) associated with his marathon.[1] With the acute and symptomatic nature of his episode, it was important to enact a brief period of relatively rapid increase of SNa concentration of approximately 4–6 mEq/L over the first 1–2 hours and then follow that with a slower increase over the following days.

HYPONATREMIA DISCUSSION

General

Hyponatremia is defined as a SNa concentration of less than 135 mEq/L and is considered severe when below 125 mEq/L. This is a common electrolyte disorder in the hospitalized population. People of any gender or age can be affected, but it may be most significant or more frequent in the elderly as they are more likely to have comorbid conditions and/or multiple medications.

Flow chart. 1: Types of hyponatremia



Pathophysiology and Causes

True hyponatremia is associated with low plasma osmolarity and therefore, this should be assessed as well. A normal or high osmolarity with a low SNa is evidence of either redistributive hyponatremia or pseudohyponatremia. These are related to excessively high levels of glucose, mannitol, triglycerides, or proteins in the serum. True hyponatremia (i.e. with low plasma osmolarity) can then be divided into three subtypes: (a) hypovolemic, (b) euvolemic and (c) hypervolemic. These are based on physical examination assessment of volume status and may prove beneficial in directing the physician to the cause of the hyponatremia. Since the ultimate treatment may depend on the underlying cause, a hyponatremia algorithm can be used to systematically evaluate the likely cause.[2]

In general, the possible causes can be narrowed down by assessing the volume status, although if there is more than one compounding factor then this can be more difficult. The most distinctive subtype is likely hypervolemic hyponatremia. Examples include nephrotic syndrome, congestive heart failure, cirrhosis and chronic renal failure. In general, these disorders lead to a volume overloaded state with much of the volume located within either the venous system or the interstitial space. This causes an apparent volume deficiency in the arterial network (i.e. decreased effective arterial volume) leading to activation of the renin-angiotensin-aldosterone and vasopressin [antidiuretic hormone (ADH)] systems. The abnormal activation of these systems leads to inaccurate regulation of the salt and water balance. On the opposite end of the volume status is hypovolemic hyponatremia. This occurs when there is loss of both water and sodium, but the extent of the sodium loss is greater. This sodium loss can occur either via two mechanisms: renal or extrarenal. These are distinguished by the urine sodium concentration. Renal losses cause the urine sodium concentration to be more than 30 mmol/L, while extrarenal losses are associated with concentrations of less than 30 mmol/L. Renal losses may result from cerebral salt wasting (CSW) syndrome, diuretics or mineralocorticoid deficiency. Extrarenal causes may include vomiting, diarrhea, excessive sweating and heat-related illness, or third-spacing from burns, peritonitis, pancreatitis, or other causes. In general, the low-vascular volume of these disorders leads to fluid retention and thirst, which can lead to dilution of the SNa. Additionally, CSW syndrome may involve various natriuretic peptides that will also decrease SNa. The final subtype is euvolemic hyponatremia, in which the patient does not show sign of either hypervolemia or hypovolemia. This can be due to several mechanisms which can be distinguished to some extent by urine osmolarity or urine sodium concentration. If urine sodium is less than

20–30 mEq/L or urine osmolarity less than 100 mOsm/L, this indicates a normal ability for the kidneys to produce dilute urine. This is most often the case with excessive hypotonic fluid intake (i.e. primary polydipsia) or even a reset osmostat. If the urine sodium and osmolarity are more concentrated, this suggests an inability to adequately dilute the urine and therefore predicts a nonosmotic release of ADH. This may be the case in hypothyroidism, hypopituitarism or adrenal insufficiency leading to glucocorticoid deficiency, or the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Note that SIADH is a diagnosis of exclusion, so you must determine that the hyponatremia is of the euvolemic type with concentrated urine and no evidence of thyroid or adrenal dysfunction. Furthermore, SIADH may be idiopathic or due to a variety of neurologic or pulmonary disorders, so further evaluation for the cause of SIADH should be instituted. One last cause of hyponatremia that should be evaluated in all patients is iatrogenically-induced via medications. Due to the different mechanisms of action of the variety of medications, it may occur with any volume status, although euvolemic hyponatremia is the most common. Evaluation of patient medications should occur as part of the workup of all hyponatremia patients.

Assessment

Since the volume status may be important in determining the cause of the hyponatremia, it is necessary to assess the volume status as part of the physical examination. Examples of hypovolemic evidence may be diminished skin turgor, dry mucous membranes, tachycardia or orthostasis. Hypervolemia may be suggested by peripheral edema, increased jugular venous distention, ascites, pulmonary rales or an S3 gallop. Lack of any of these physical findings is consistent with euvolemia. Outside of the physical examination findings, a good patient history and some laboratory tests will help to determine the ultimate cause. Much of the time, the hyponatremia is found incidentally upon routine laboratory blood work. This often suggests a chronic nature of the disease and usually has no associated symptoms. In an acute setting, symptoms are more likely to develop and the severity depends on the extent of the hyponatremia (*Source*: 5MinuteConsult). Initial symptoms may include nausea, vomiting, and malaise which may proceed further to headache, lethargy, confusion and decreased awareness or responsiveness. If very severe or rapid decreases in sodium occur then seizure, coma, or respiratory arrest may result. Recall that the underlying physiology of the symptoms is due to the fluid shift from extracellular to intracellular spaces, which primarily affects the brain in the rigid cranium, thus leading to primarily neurologic symptoms.

Besides symptoms of the hyponatremia itself, some patients may exhibit symptoms of the underlying cause of the hyponatremia. For example, a patient with incidental hyponatremia may also have weight gain, fatigue, constipation, and sensitivity to cold which could suggest hypothyroidism as the underlying cause for the hyponatremia. Whether or not symptoms are present, various laboratory analyses may be helpful in determining the diagnosis. As previously mentioned, SNa, plasma osmolarity and volume status must be assessed to determine the specific subtype of hyponatremia (i.e. hypo-, hyper- or euvolemic). If indicated, additional laboratory tests may include urine sodium and osmolarity, renal function, hepatic function, thyroid function, adrenal function, serum glucose and lipids. Furthermore, if SIADH is ultimately diagnosed then a legitimate effort should occur to rule out underlying CNS or respiratory causes, which may require chest X-ray and/or head CT.

Treatment

The mainstay of treatment for hyponatremia is correction of the serum sodium abnormality. This must be tailored to the nature of the hyponatremia, whether or not symptoms are present and any underlying pathology. For asymptomatic patients, correction of the sodium deficit should be less aggressive. In hypovolemic patients, isotonic saline should be used to replenish the intravascular volume. This should inhibit the further release of ADH. With hypervolemia, additional fluids are not indicated, and instead the patient should be treated with salt and fluid restriction (< 1L/day) and loop diuretics. A vasopressin-2 (V2) receptor antagonist (i.e. conivaptan or tolvaptan) may be considered to decrease the effect of the inappropriate release of ADH. Furthermore, it is very important to provide treatment for the underlying disease, as this is the ultimate cause for the hyponatremia in the first place. Lastly, in euvolemic patients, fluid restriction is used as well. If fluid restriction of less than 1L/day does not improve the hyponatremia, it may be necessary to further restrict fluids or add a V2 receptor antagonist to aid in the correction. In all of these patients, correction of the serum sodium should not exceed 8–12 mEq/L/day.

Acutely symptomatic patients (i.e. neurologic deficit) should undergo an initial brief period of relatively quick correction of the sodium deficit to reverse the symptoms. This brief (2–3 hours) intervention can utilize either hypertonic saline alone or isotonic saline with a loop diuretic. Both of these actions intend to provide relatively more sodium compared to water for a more rapid reversal of symptoms. During this period, correction should be approximately 2 mEq/L/hr until the symptoms resolve. Then the previously described treatment for asymptomatic patients can be

used. Symptomatic patients with chronic hyponatremia represent the population that is most difficult to treat. Although all patients should be corrected conservatively, these patients are most likely to encounter severe side effects from correction that is too rapid. Sodium correction should be limited to 0.5–1 mEq/L/hr and not to exceed approximately 8–10 mEq/L/day. Patients with chronic hyponatremia have had time to compensate for the fluid shifts, and this represents higher risk for neurologic damage by adding more extracellular sodium. This could lead to cell shrinkage from fluid outflow potentially resulting in central pontine myelinolysis (CPM) and/or extrapontine myelinolysis (EPM). These can lead to irreversible neurological damage. In contrast, acute hyponatremic patients have not had time to compensate for the hyponatremia and therefore are less likely to be harmed by the rapid fluid outflow from the cells and can tolerate a relatively more rapid correction.

REFERENCES

1. Howard D. Backer, David R. Shlim. (2011). Problems with Heat & Cold. Centers for Disease Control and Prevention. [online] Available from http://wwwnc.cdc.gov/ travel/yellowbook/2012/chapter-2-the-pre-travel-consultation/problems -with-heat-and-cold.htm. [Accessed April 2nd, 2013]
2. Lien YH, Shapiro JI. Hyponatremia: clinical diagnosis and management. Am J Med. 2007;120(8):653-8

Case Study 96: Hyperkalemia

CASE HISTORY

A 60-year-old diabetic patient is presented to the emergency department with the chief complaint of feeling weak, tired, confused for the past 24 hours. He also has hypertension for which he is taking lisinopril and spironolactone. On examination, his vitals are stable. He also complains of nausea, vomiting, diarrhea, abdominal pain, paresthesia such as tingling and numbness, muscle weakness, flaccid paralysis, tachypnea due to respiratory muscle weakness and bradycardia.

LABORATORY TESTS

Laboratory tests ordered for basic metabolic profile, complete blood count (CBC) and arterial blood gas (ABG) (Fig. 1).

ELECTROCARDIOGRAM

Electrocardiogram (ECG) shows complete heart block, peaked T waves, prolonged PR intervals and widened QRS complexes (Fig. 2).

CAUSES OF HYPERKALEMIA

Hyperkalemia is defined as serum K^+ greater than 5.5 mEq/L and can lead to life-threatening consequences.

Fig. 1: Toxic Levels of Hyperkalemia

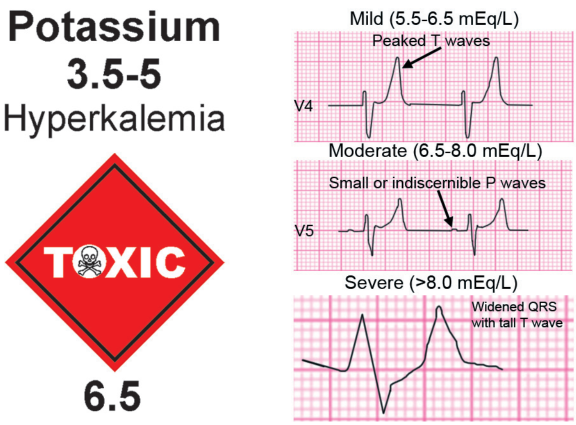


Fig. 2: EKG Changes
Source: wikimedia.com

Pseudohyperkalemia has been reported in as high as 20% of blood samples with elevated potassium levels. Traumatic venipuncture is the most common cause of traumatic hemolysis. Potassium release from muscles usually occurs distal to tourniquet placement. Lastly, when blood clots form in the specimen tube, potassium is released from cells; this usually occurs in the presence of leukocytosis or thrombocytosis. Therefore, if hyperkalemia is present in an asymptomatic patient, it is vital to repeat with a new blood sample.

If repeat testing confirms hyperkalemia then the source of hyperkalemia needs to be determined. Urine potassium can be helpful to differentiate between potassium release from cells or decreased renal potassium excretion. Urine potassium greater than 30 mEq/L suggests a transcellular shift and lower urine potassium excretion suggests impaired renal excretion.

Hyperkalemia is often associated with acidosis; however, no evidence demonstrates acidosis as a cause of hyperkalemia. Metabolic acidosis and hyperkalemia are often present together in conditions such as renal failure and renal tubular acidosis secondary to impaired renal excretion of potassium. Rhabdomyolysis in association with impaired renal clearance can also lead to hyperkalemia, since the kidneys are unable to clear the increased amount of potassium released into the extracellular space by muscle cells.

Drugs can also be a cause for hyperkalemia, therefore it is important to review medication history in patients with hyperkalemia. Beta receptor antagonists and digitalis are most commonly associated with hyperkalemia resulting from a transcellular shift. Digitalis toxicity can result in severely high potassium levels, which can lead to significant cardiac

consequences. Several more drugs are associated with hyperkalemia secondary to decreased excretion such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), potassium sparing diuretics and nonsteroidal anti-inflammatory drugs (NSAIDs). They impair excretion by inhibiting the renin-angiotensin-aldosterone system.

Renal and adrenal insufficiency can result in hyperkalemia. This should be suspected if the patient's glomerular filtration rate (GFR) is below 10 mL/min or urine output is lower than 1 L/day.

CLINICAL MANIFESTATIONS

The most concerning manifestation of hyperkalemia is slowing of electrical conduction in the heart. The effect on the electrical conduction depends on the potassium serum level. Figure 3 shows ECG changes associated with progressive hyperkalemia.

"Peaked T-waves" are usually the first ECG manifestations in hyperkalemia. These are usually appreciated on precordial leads V2 and V3. Progressive hyperkalemia causes decrease in the P-wave amplitude and progressive increase in the PR interval leading to first-degree heart block. Eventually, QRS duration is increased and P waves disappear resulting in complete heart block seen at serum K^+ levels greater than 10 mEq/L. The final event seen at serum K^+ levels of 14 mEq/L is ventricular asystole.

"Peaked T-waves" are often observed in patients with metabolic acidosis.

TREATMENT

The treatment plan of hyperkalemia using cardiac monitor is described in Flow chart 1.

Following are the additional treatment options:

- Nebulized albuterol 2.5 mg in 3 mL normal saline (NS) every (q) 20 minutes
- Sodium bicarbonate 1 ampule 50 mEq in 5 minutes
- Furosemide 20–40 mg intravenous pyelogram (IVP).

Note: Treat the underlying cause, if it is secondary to Addisonian crisis; give steroids. Antigen-binding fragments (Fab) are given for digoxin toxicity. If digoxin toxicity as well as hyperkalemia coexist, never give calcium gluconate/calcium chloride.

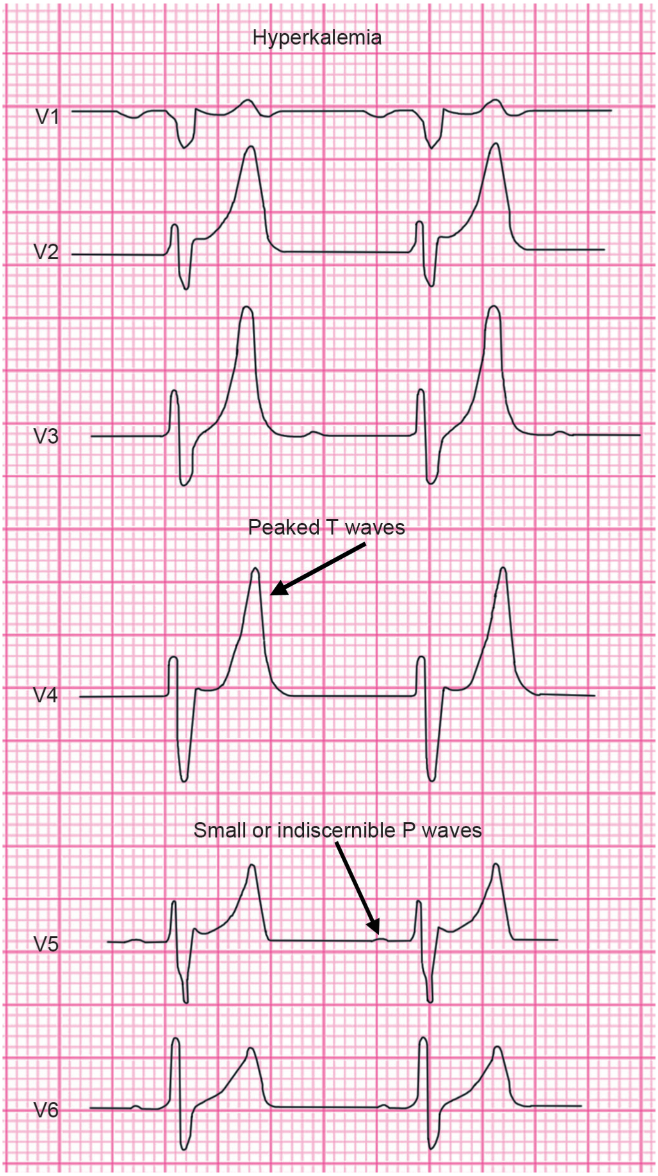
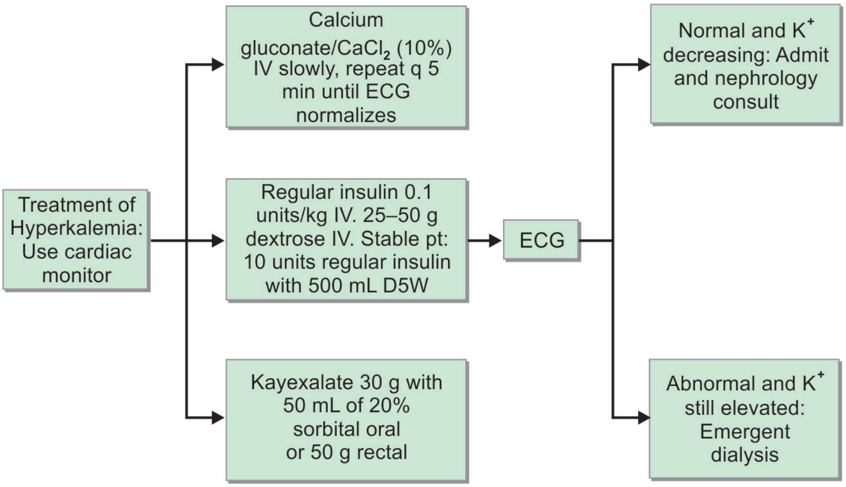


Fig. 3: EKG Changes
Associated with Hyperkalemia
Source: wikimedia.org

Flow chart 1: Treatment of hyperkalemia



Source: Badar M Zaheer, MD

ADDITIONAL READING

1. High Potassium Levels 2013 National Institute of Health http://www.nlm.nih.gov/medlineplus/ency/article/001179.htm
2. EKG Changes with Hyperkalemia www.wikimedia.org

Case Study 97: Hypomagnesemia

CASE HISTORY

A 45-year-old male is brought to the emergency room for the chief complaint of muscle weakness, confusion, decreased reflexes, jerky movements, high blood pressure and irregular heart rhythms. He has been drinking heavily but not eating. He is a known hypertensive and takes hydrochlorothiazide (HCTZ) 25 mg daily.

DISCUSSION

Hypomagnesemia is commonly observed in hospitalized patients and has been reported in as high as 65% of patients in the intensive care unit (ICU). Therefore, it is vital to recognize conditions that predispose a patient to hypomagnesemia (Flow chart 1) and closely monitor their serum magnesium levels.

Aminoglycosides, amphotericin and pentamidine have been shown to cause hypomagnesemia. Aminoglycosides have been reported to cause hypomagnesemia in approximately 30% of patients by blocking the reabsorption of magnesium in the ascending limb of the loop of Henle.

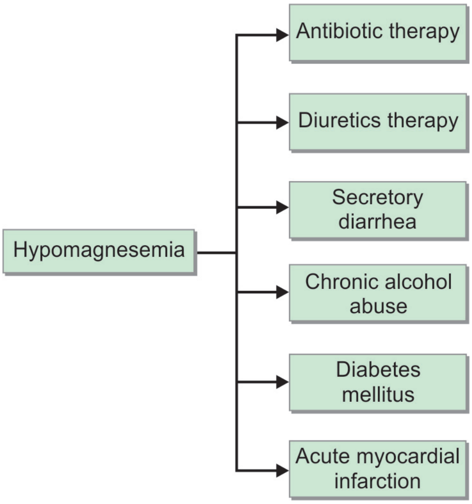
Diuretic therapy, especially loop diuretics are most often associated with drug-induced hypomagnesemia They interfere with magnesium reabsorption while inhibiting sodium reabsorption. Fifty percent of patients on chronic diuretic therapy with furosemide are found to have decreased magnesium levels. Thiazide diuretics, on the other hand, are only associated with hypomagnesemia in elderly patients.

The most common cause of hypomagnesemia in the US is alchohalism. Patients with chronic alcohol abuse are at an increased risk for hypomagnesemia secondary to poor nutrition and chronic diarrhea. Since magnesium is required in the conversion of thiamine to thiamine pyrophosphate, it is often associated with thiamine deficiency. Magnesium levels should be closely monitored in patients on thiamine supplementation.

Secretory diarrhea predisposes a patient to hypomagnesemia since lower gastrointestinal (GI) excretions consist of high concentrations of magnesium (10–14 mEq/L). Upper GI excretions, on the other hand, are lower in concentration of magnesium.

Hypomagnesemia is also commonly seen in patients with insulin-dependent diabetes secondary to urinary magnesium loss accompanied with glycosuria. Additionally, up to 80% of patients with an acute myocardial infarction have been found to have hypomagnesemia in

Flow chart 1: Causes of hypomagnesemia



the first 48 hours. This decrease in magnesium serum levels has been hypothesized to be secondary to endogenous catecholamine excess.

CLINICAL MANIFESTATIONS

Hypomagnesemia is often accompanied with associated electrolyte abnormalities such as hypokalemia, hypophosphatemia and hypocalcemia.

Emergency Room Care and Disposition

1. Correct volume deficits and any associated potassium, calcium, or phosphate deficiencies.
2. In alcoholic patients, who have delirium tremens (DT), or pending DTs give, 2 grams of magnesium sulphate in the first hour, and then 6 grams in the first 24 hours. Check for DT's every 15 minutes until the serum magnesium levels come back to 3. 5 mEq/L or above.

ADDITIONAL READING

1. Hypomagnesia 2013 National Inisitute of Health
http://www.nlm.nih.gov/medlineplus/ency/article/000315.htm

CHAPTER

20

Ocular Emergencies

Case Study 98: Red Eye

CASE HISTORY

A 20-year-old male is brought to a rural emergency room by his mother. The man has a bandana wrapped around his eyes. He is placed in an examination room, and his mother quickly turns off the lights until the nurse arrives. Upon arrival to the room, the mother tells the nurse that light hurts her son's eyes. The patient has redness and pain in his right eye that started 2 days ago. There is no drainage and his mother, who has checked his eyes, claims there is nothing inside them. The patient states he has had a loss in focus and clearness of vision in his right eye for the past 2 days. The patient's medical history has nothing notable. Upon examination, the eyelids and lashes are normal. Some ciliary congestion is seen in the sclera on the right eye, but not the left. The patient experiences 8/10 pain when the pupils are examined with light. The right pupil is smaller, irregularly shaped and less reactive than the left pupil to light. The doctor gives a few drops of tetracaine with no relief. Fluorescein uptake is negative. The anterior-chamber depth is narrow, and the slit-lamp examination is positive for cell and flare. Diagnosis by the emergency department (ED) physician is acute iritis, and long-acting cycloplegic topical medicine is given for the ciliary spasm. Upon consultation with the ophthalmologist, a prescription for a topical steroid is written. A follow-up appointment is arranged with the ophthalmologist within 24 hours.

DISCUSSION

Table 1 discusses symptoms and diagnosis of red eye.

Table 1: Symptoms and diagnosis of red eye

	Symptoms	*Diagnosis*
Conjunctivitis (Fig. 1)	Red eye + gritty foreign body + discharge	Red eye without change in vision or pain
Subconjunctival hemorrhage (Fig. 2)	Blood between the conjunctiva and the sclera	Hemorrhage seen after trauma; may be spontaneous or related to systemic injury
Corneal abrasion (Fig. 3)	Pain + foreign body + tearing + photophobia	Abrasions, usually the result of trauma; patient may or may not remember the event. Patient may have blurry vision if abrasion is big enough/covers more of the eye
Acute uveitis (Fig. 4)	Severe photophobia + red eye + blurry vision + irregular pupil	Inflammation of the uveal tract. Acute iritis is the most common.

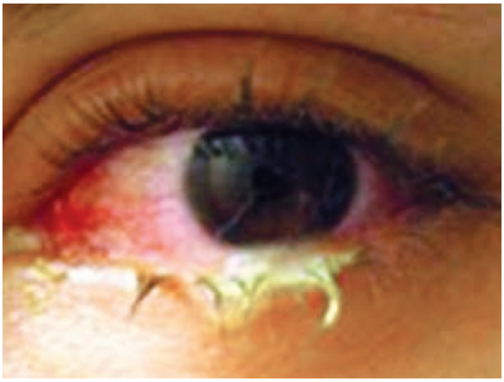


Fig. 1: Conjunctivitis

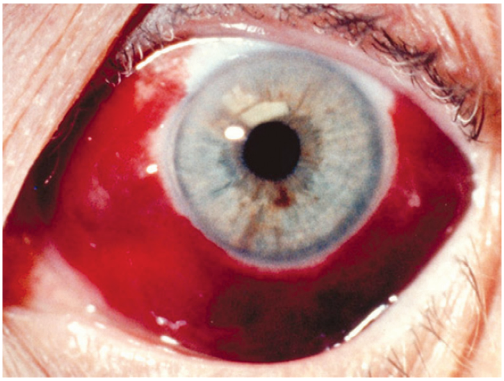


Fig. 2: Subconjunctival hemorrhage

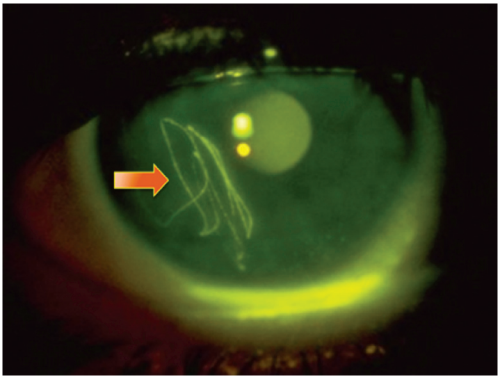


Fig. 3: Corneal abrasion

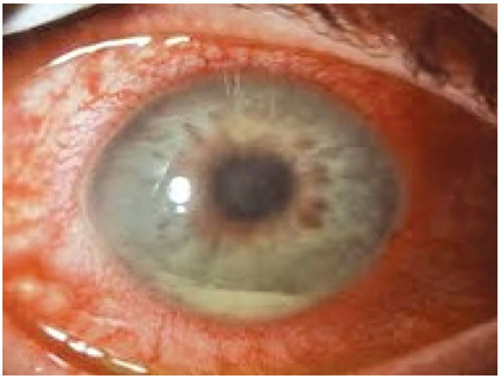


Fig. 4: Acute uveitis

CAUSES

The possible causes of acute uveitis in patients include infections like tuberculosis, herpes simplex, herpes zoster, toxoplasmosis, cytomegalovirus, or syphilis. Autoimmune, idiopathic diseases, and trauma are other possible causes.

Few things you should never miss in the eye:

- Acute glaucoma
- Temporal arteritis
- Anterior uveitis (iritis)
- Central retinal artery occlusion
- Retinal detachment.

TREATMENT

The treatment of acute uveitis involves topical corticosteroids. This treatment involves risk of development of glaucoma, cataracts, reactivation of herpes and thus an ophthalmologist must be consulted immediately.

Case Study 99: Acute Angle Closure Glaucoma

CASE HISTORY

A 65-year-old woman presents to emergency room (ER) with right eye pain, redness, severe headache, and blurred vision for the past 4 hours after painting picture for several hours. The pain is progressively worse. The left eye feels normal and has no problem with vision. She accidentally injured her head 1 week ago and she believes this may be the cause. She denies any photophobia, tearing or discharge. There is no similar eye pain or blurring vision in the past. She only wears glasses and is far sighted. When seen in the ER, she sees halos around her eye.

Past Medical History

No medical problem was observed and no use of any medication was found.

Past Surgical History

Negative.

PHYSICAL EXAMINATION

On examination the patient has visual acuity of 20/30 in the left eye. Visual acuity is finger counting in the right eye. Visual field is within normal limits. Gentle palpitations of closed right eye reveal that it is more harder than left eye. Her left pupil is 5 mm fixed and unreactive. Her left eye appears normal. Size of pupil is 3 mm and brisk reaction is present. Extraocular movements are intact and painless. Right cornea is slightly cloudy and because of the cloudy cornea, funduscopy is impossible. The left fundus appears normal; temporal arteries are nontender and they are pulsatile. The rest of physical examination is normal.

DIAGNOSTIC TESTS

Slit-lamp examination measured an intraocular pressure of 52 mm Hg in the right eye and normal pressure in the left eye. The pressure was found using Tono-Pen. Slit-lamp examination also revealed narrow anterior chambers.

DISCUSSION

Acute close angle glaucoma commonly presents with:
- Headache
- Halo around vision

- Severe eye pain with redness of the eye
- Nausea and vomiting.

DIFFERENTIAL DIAGNOSIS

- Anterior uveitis
- Conjunctivitis
- Corneal ulcer
- Traumatic endophthalmitis.

PATHOLOGICAL FINDINGS

- Atrophy and cupping of optic nerve.
- Loss of retinal ganglion cells and their axons produces defects in the retinal nerve fiber layer.
- Funduscopic examination shows optic nerve cupping.

TREATMENT PLAN

Treatment goal is to lower intraocular pressure with an ophthalmology consult. Carbonic anhydrase inhibitor, acetazolamide (Diamox®) 250 mg po (per os or by mouth) qid (four times a day) for 6 hours can be given orally or locally. Also, osmotic agents such as mannitol are used to dehydrate the vitreous humor and this reduces intraocular pressure.

PRACTICE PEARL AND LESSON TO LEARN

Never ever in your life miss a case of Glaucoma , Acute Uveitis, Temporal arteritis and other causes of sudden loss of vision. Consider every red eye and headache a potential for these diagnoses. For malpractice law suits, missing diagnosis makes up for about 70% of claims.

ADDITIONAL READING

1. National Institute of Health: http://www.nlm.nih.gov/medlineplus/ency/article/000054.htm
2. Emedicine: http://www.emedicinehealth.com/subconjunctival_hemorrhage_bleeding_in_eye/page12_em.htm
3. Wikipedia: http://en.wikipedia.org/wiki/Subconjunctival_hemorrhage

Case Study 100: Central Retinal Artery Occlusion

CASE HISTORY

A 68-year-old woman is presented to the emergency department (ED) with the chief complaint of sudden loss of vision in the left eye. She was eating dinner with her family after gardening when she experienced the vision loss. She denies nausea, vomiting or eye pain. She has a history of hypertension and atrial fibrillation. She is not diabetic.

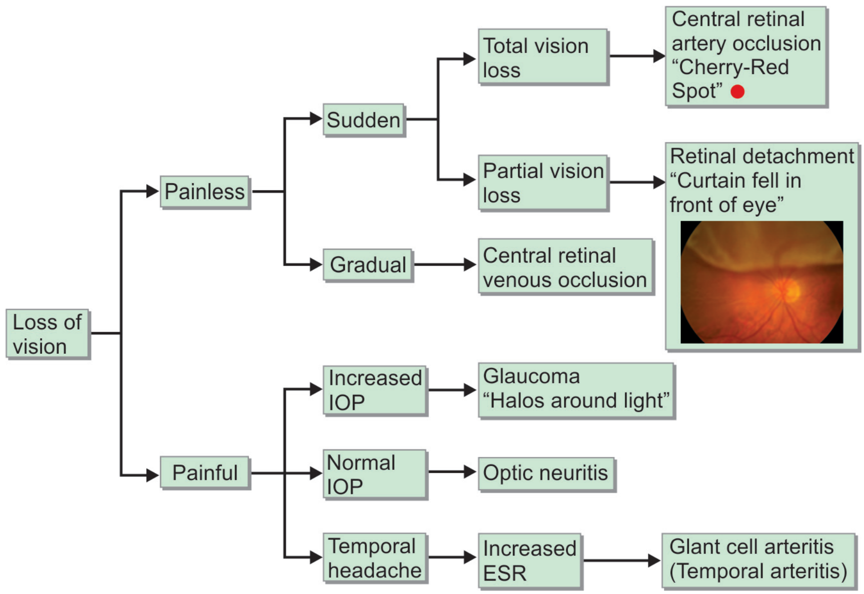
PHYSICAL EXAMINATION

On examination, she has complete loss of vision in the left eye. Right eye vision is 20/40. Pupils are equal in size and reactive to light. Sclera and intraocular pressure in both eyes are normal. Left eye shows pale fundus with cherry-red spot.

ASSESSMENT AND PLAN

The diagnosis is central retinal artery occlusion. The differential diagnoses of vision loss can be visualized in Flow chart 1.

Flow chart 1: Differential diagnosis of vision loss



In the ED, the management goal was to dilate the artery to minimize further ischemic injury to the retina and to refer to ophthalmology for advanced treatment.

- Timolol 0.5% eye drops
- Intravenous acetazolamide 5 mg
- Patient was instructed to hyperventilate into a paper bag during the encounter to increase her arterial CO_2 in attempt to further dilate the affected artery.
- Intermittent, 5-second digital massage of the entire globe was performed by both physician and patient for 15 minutes.

ADDITIONAL READING

1. National Institute of Health: http://www.nlm.nih.gov/medlineplus/ency/article/001028.htm
2. Emedicine: http://emedicine.medscape.com/article/1223625-overview

CHAPTER

21

Disaster Management

Case Study 101: Mass Casualty—Train Accident

CASE HISTORY

In 1999, a semi truck driving down the highway pulled in front of an Amtrak train in Kankakee, Illinois, USA. The train was derailed and crashed causing 40 tragedies, 122 injuries and 11 deaths (Fig. 1).

For this scenario let us imagine that a passenger train carrying 110 passengers is travelling down the track on a hot summer night when it collides with a tanker train hauling highly flammable liquids. The commercial train is operated by a small crew of eight members. The collision occurs at night and when the trains collide, one of the tankers bursts into flames and two more cars simply rupture and their contents

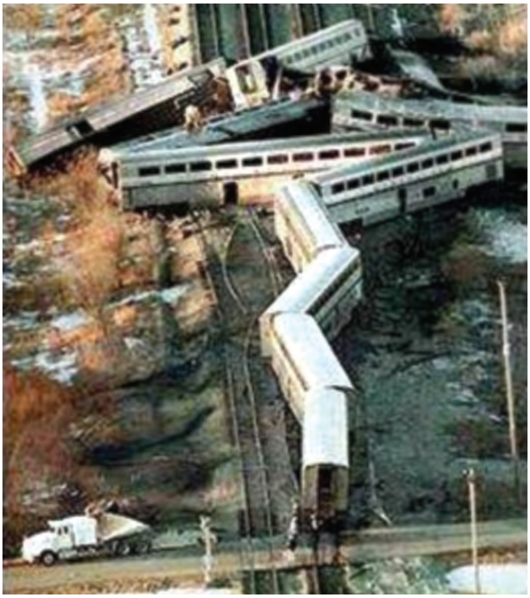


Fig. 1: Train accident in Kankakee, Illinois, USA in 1999
Courtesy: Wikimedia.org

go everywhere. After the collision, emergency teams arrive on the scene and do an initial assessment. There are 70 injuries in total with 20 deaths including two firemen who died trying to put out the fire. The paramedics who arrived on the scene start to triage out the victims and come up with 20 Category Red patients (12 with extensive second- and third-degree burns), 19 Category Yellow patients (5 with second-degree burns), 25 Category Green patients (5 with limb deformities), and 6 Category Blue patients (4 with catastrophic third-degree burns). The train crash occurred within 12 miles of a major hospital with 35 open beds. In 50 miles there is a hospital with a major trauma center that has a dedicated burn unit.

DISCUSSION

In disaster situations, there are many factors that need to be taken into consideration in a very short period of time. The first responders will often set-up a triage system as illustrated in the case example (Fig. 2). Color coding victims is an easy way to determine which patients need to receive treatment first and which patients can wait till others are done. In this situation, Red injury is for a life-threatening injury that requires immediate attention or operation. Yellow injuries are ones that may become life (or limb) threatening if care is not given within a couple hours.

Fig. 2A

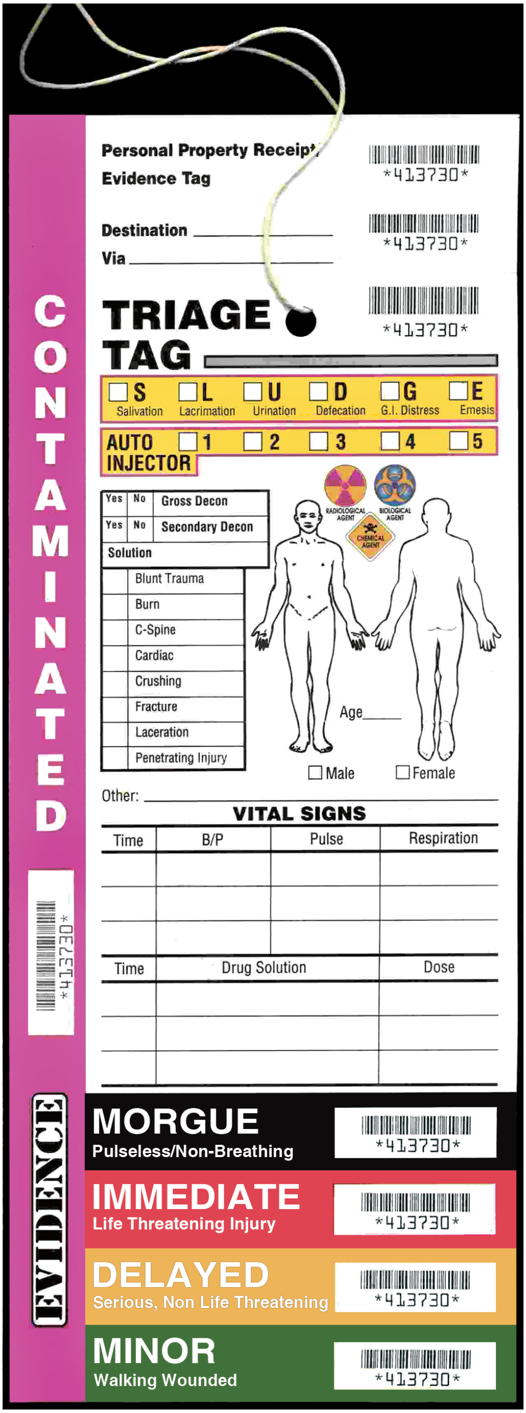


Fig. 2B: Triage systems

Courtesy: www.thevestguy.com and wikimedia.org

Green patients are considered the walking-wounded; these patients have suffered only minor injuries and may be asked to get up and walk away from the disaster area by themselves (note this is also a good way to find Green label patients). Lastly, patients listed as black were dead when the primary triage went through the casualties. Sometimes, triage units list patients as Blue, or severely injured. The prognosis of these patients depends on the current number of casualties to receive care (Red and Yellow patients). Blue patients are often given palliative care hoping they survive until patients with a better probability of surviving make it to the hospital, and then they will be taken.

A situation like this may be considered as mass casualty incident (MCI), a situation where personnel and equipment are overwhelmed. This includes emergency responders to the scene of the accident as well as local hospitals and hospital staff. In this scenario there is a local hospital within 25 miles of the scene with 35 open beds, but the next hospital is 50 miles away. Emergency medical teams will need to set up alternative areas to stabilize and treat victims until emergency medical vehicles arrive and hospital beds can be found for all injured in the accident. These stabilization areas should be accessible to emergency medical vehicles. Caution should be taken, and these areas should be far enough from the disaster area as not to cause further injury (chemical, mechanical, or otherwise) to the patients within those areas. Typically, as patients are brought into the stabilization area they are triaged again to assess severity of conditions. They should then be moved to the appropriate area of the stabilization zone to receive treatment and await extraction.

Once all hospital-bound patients have made it on an ambulance, the disaster crew will come in and start the cleanup on the area. Part of this cleanup involves dealing with the casualties from the disaster and getting those bodies to the morgue.

PRACTICE PEARL

Remember! In the event of electrical injury, it is never a good idea to put low priority on initial pulseless patients. If a group of persons is struck by lightening, attention should be directed to those with no signs of life (black tagged), because the others will probably recover. Immediate CPR and prevention of anoxic death are essential.

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